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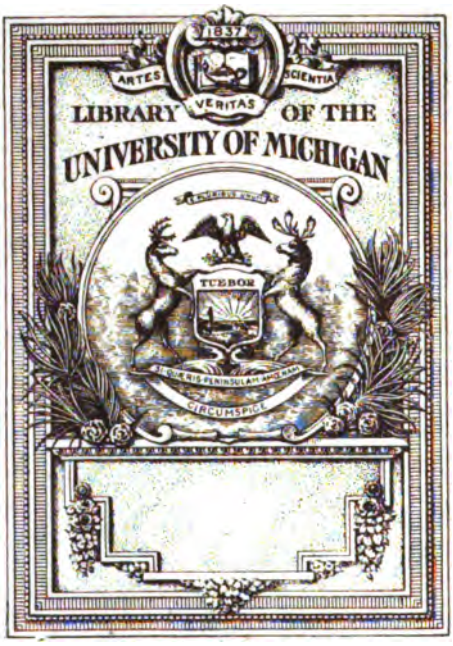
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For 1879.

Presented to both Houses of Parliament by Command of Her Majesty.

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PUBLIC HEALTH.

ANNUAL REPORT

OF THE

MEDICAL OFFICER

OF

THE LOCAL GOVERNMENT BOARD

FOR THE YEAR

1879.

P 761. Wt. 15707.

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REPORT.

TO THE RIGHT HONOURABLE JOHN GEORGE DODSON, M.P., PRESIDENT OF THE LOCAL GOVERNMENT BOARD, &c., &c.

SIR,

MEDICAL OFFICER'S REPORT.

IN supplement to the Board's Ninth Annual Report, I have now the honour to lay before you my report of the special work done through the Medical Department during 1879 in matters concerning the public health.

The year has been memorable in the Medical Department by the retirement of its principal officer, Dr. Seaton, owing to ill health, and by his death shortly after his retirement. To the words of esteem and regret for Dr. Seaton contained in the Board's Report, his immediate colleagues, who can no longer profit by his knowledge and judgment, and who have lost his example of accuracy and earnestness, claim to add their more particular expression of regard and of sorrow.

The present Report has reference in the main to the proceedings of the Medical Department during Dr. Seaton's tenure of office.

VACCINATION.

I. (*a.*) Statistics as to the work of the Department in supervision and in promotion of vaccination will be found in Appendix A., Nos. 1, 2, and 3. The digest of Vaccination Officers' Returns shows the percentage of children accounted for* as to their vaccination, among those born in England and Wales during 1877, to have been, to a trivial degree, smaller than in 1876, and the difference between the two years to be most apparent in the Metropolitan returns. Of children born in 1877 in Metropolitan Unions, 7·1 per cent. had not been accounted for at the time the returns were made. In extra-metropolitan Unions this percentage was 4·1 only. The investigation which the Board set on foot during 1878 into the significance of the larger metropolitan figures has recently led to some improvements in London arrangements, but the result of these cannot of course be yet announced.

The actual working of arrangements for public vaccination was in 1879 examined by the Board's Medical Inspectors in 348 unions, the examination extending to 1,616 vaccination districts held by 1,520 public vaccinators. The work of 76 per cent. of

* That is, in four divisions, as follows: (1) successfully vaccinated, (2) dead without vaccination, (3) having had small-pox before vaccination, and (4) the so-called insusceptible. The successfully vaccinated of 1877 numbered 766,824, or 86·3 per cent. of the births, against 763,277, or 86·0 of the births of 1876.

MEDICAL OFFICER'S REPORT.

these vaccinators was found to be of first class excellence, and awards of money from the Parliamentary Grant were made to 60 per cent. of the 1,520, who, having been in office for a year and upwards, had in all respects exactly fulfilled their duties. These percentages show a satisfactory advance on the corresponding figures of previous years.

(*b.*) The National Vaccine Establishment continued during the year its operations for the collection, examination, and distribution of vaccine lymph to medical practitioners; and 28,419 charges of lymph were supplied by it to 9,410 applicants. The precautions adopted for securing efficiency and safety in the lymph supply have been continued with results so precisely identical with those of the previous year that I need not repeat the particulars given in the Report for 1878.

The purpose of the National Vaccine Establishment is, as many medical practitioners need to be reminded, not the provision of indefinite supplies of lymph for the vaccination of each person who may require vaccination in a district or a medical practice, but the supply of such amount of lymph as shall suffice for the establishment of local "stocks," and for the replacement by lymph of a new stock and of undoubted efficiency, of any local stocks that may be found to be giving unsatisfactory results.

Having this purpose more especially in view, and desiring always to know whether there was any better lymph to be had for the use of the Establishment than that which is selected from among the supplies furnished by the experienced public vaccinators who form the staff of the Establishment, the Board caused examination to be made of the effects upon children of lymph which had been derived from the Animal Vaccine Institution of Brussels. Dr. Cory, of St. Thomas's Hospital, who officiates at the chief educational Vaccination Station of the metropolis, undertook this investigation for the Board, and his Report forms Appendix A., No. 4, to this report. It will suffice in this place to say of Dr. Cory's results with Dr. Warlomont's lymph that in some respects they differed from those which he habitually obtains with long-humanized lymph; that there was no reason for preferring the results of the calf lymph; and that the humanized lymph had, in its arm-to arm transmissions, the advantage over the calf lymph of showing greater uniformity of action.

Reference will presently be made to some experimental studies by Dr. Klein into the pathological relations of cow-pox and small-pox.

REPORTS OF MEDICAL OFFICERS OF HEALTH.

II. The principal reports received by the Board from Medical Officers of Health for urban and rural sanitary districts of the kingdom have been read in the Medical Department. They have related to the health of the districts during the year 1878,

and to various pecial occurrences of disease in 1879. Of the annual reports I can only repeat what was said last year, that they show every grade of care about sanitary matters on the part of Health Officers and Sanitary Authorities, with strong indications that judgment and zeal on the part of the Medical Officers have a considerable share in determining the activity of those authorities. In some measure, no doubt, owing to want of skill in reporting, but also in a measure owing to want of observation of circumstances affecting health or to insufficient apprehension of the significance of what was observed, too many instances are found where the reports of Medical Officers of Health fail of giving an adequate representation of the sanitary state of their districts, while the advice conveyed in them is in a corresponding degree unsatisfactory. But for this state of matters it is often too evident that the authorities themselves are in a measure responsible. They have often selected as their Officers of Health gentlemen with no better qualification for the appointment than the tenure of some other medical office; or they have, in the performance of their sanitary functions, taken no counsel with their Officers of Health, and have thus encouraged perfunctory work and perfunctory reporting. Again, as regards special occurrences of infectious disease, the reports required by the Order of the Board have too often not been sent, and the Board has been left without information of serious outbreaks of disease until it learned of them from private individuals, or until a serious mortality had resulted. It is satisfactory to turn from this necessary criticism of the work of some officers of health to record the distinct proof of progress afforded by the reports of many others; of progress in the officers' power of research, in the value and importance of their advice, and in the trust in which they are held by their sanitary authorities. Thus the impression conveyed by the body of reports, annual and special, which have been examined in the Medical Department, has been that throughout the kingdom the promotion of health and the prevention of disease are each year receiving wider and more intelligent interest from local communities.

LOCAL INQUIRIES AS TO PREVALENCE OF DISEASE.

III. Thirty local inquiries by the Board's Medical Inspectors have been made during the year. They have had reference to the areas of jurisdiction of 38 sanitary authorities. These numbers are larger than in the preceding year. The occasion for the inquiries arose sometimes from local representations, but more often from the evidence afforded by the Registrar General's Quarterly Returns of some exceptional prevalence of disease in the several localities. In undertaking this larger number of local inquiries the Board had temporary assistance from five gentlemen engaged for special duty during the illness of Dr. Seaton, and during the exceptional occupation of members of its permanent medical

MEDICAL OFFICER'S REPORT.

staff. An abstract of the results of the year's inspections will be found in Appendix A., No. 5. In large measure the inquiries were concerned with scarlatina, diphtheria, and enteric fever, in places where these diseases had appeared in an actively epidemic form. They showed sanitary authorities and their officers exercising various degrees of vigilance over circumstances affecting health. Too often the serious amount of disease that had appeared in the districts was demonstrably related to neglect of sanitary duty.

The reports made by the Inspectors have been of a kind to be most serviceable to authorities and officers of health; and the recommendations with which the reports were accompanied have been calculated to afford to local authorities very material assistance in the performance of their sanitary functions. At the same time, the reports have brought home for the information of the Board a variety of experiences illustrative of previous knowledge, or adding to the store, and in some instances indicative of directions for further observation and research. Under this aspect, three of the reports deserve special mention.

(*a.*) Mr. Power, examining into a remarkable outbreak of "fever" on board the Reformatory School Ship "Cornwall," was able to disprove its identity with enteric fever, and proceeding to search for direct post-mortem evidence of its nature, found himself concerned with a parasitic disease, in certain essential particulars resembling that produced in the human subject by the presence of Trichina (see App. A., No. 6). Some anatomical peculiarities of this "Cornwall" parasite (noted at the time by Mr. Power, but by him regarded as of no generic importance) tended, indeed, to throw doubt on its identity with Trichina: and a further report on the subject by Dr. Bastian (App. A., No. 7) refers the parasite to the genus Pelodera, which has not heretofore been known to invade the human body. This view adds materially to the interest and importance of Mr. Power's observations of the disease on board the "Cornwall."

(*b.*) Dr. Thorne's investigation of a strongly-marked outbreak of enteric fever at Caterham and Redhill resulted in conclusive proof of the manner of its distribution, and was of singular interest as demonstrating how limited a quantity of infective material may suffice under favouring circumstances to do wide-spread mischief (see App. A., No. 8). In one of the wells of the local water company a new adit was being formed, and among the workmen was one who during his occupation in the adit suffered from a slight attack, at the time unrecognised, of enteric fever. He, when pressed by diarrhœa, used for his relief a bucket that was provided for raising to the surface the chalk dug from his workplace underground. It was found to be certain that the well water could thus have become contaminated, and there was no suggested way of its contamination by any other means. For the rest, quite satisfactory evidence connected the supply of water from this well with the prevailing epidemic that extended

beyond Caterham to Redhill and other localities supplied by the company's water.

(*c.*) Dr. Airy, inquiring into the circumstances of a peculiarly sudden outburst of scarlet fever at Fallowfield, near Manchester, was able to trace the extension of the disease by means of milk supplied from one dairy. Though no satisfactory evidence was to be had as to the way in which the milk came to receive its infective properties, the coincidence of distribution of the milk and of the disease was such as to leave no doubt that the one was carried by means of the other. Thus the facts of the report (App. A., No. 9,) confirming some earlier experiences, afford fresh and helpful indications for further investigation into the pathology of scarlet fever.

FOREIGN EPIDEMICS.

IV. The subjects of quarantine and of Levantine plague have made considerable demands on the Medical Department during 1879. I propose to submit to you in a separate form papers relating to these matters.

AUXILIARY SCIENTIFIC INVESTIGATIONS.

V. (*a.*) In an important paper appended to this Report (App. B., No. 1) Dr. Klein records the results of his further study of the lymphatic system of men and animals. His previous investigations (Report of Med. Off. of Privy Council, No. III., New Series) had reference to the lymphatics of the serous membranes and of the lung; and were calculated to throw light on those chronic processes of infection, which, especially under the name of tubercle, are recognised as the cause of an enormous amount of disease. Dr. Klein's present researches have related to other parts of the lymphatic system, and they contribute much to a recognition of the part that is played, both in health and disease, by the several tissues that compose the skin and mucous membranes. For the pathologist, his observations are of singular interest, as indicating the methods by which particulate organisms, such as we are learning to regard as being intimately related to the contagion of acute infectious disease, can obtain access into the tissues of the body. So, from the standpoint now reached, it has become easier to understand the processes by which skin and mucous membrane, crowded with such organisms, can spread them abroad in air and water to carry infection to other living bodies.

(*b.*) Investigation into the evidences of pathological identity between small-pox and cow-pox was made by Dr. Klein during the year 1878–9. Like Gassner, Theile, and Ceely, he attempted to produce vaccinia in the calf by inoculating the animal with human variola; but hitherto his results have so far differed from theirs that in 33 instances, his experiments have failed of producing recognisable vaccinia. Yet there is nothing in these observations that can safely be regarded as disproving the

transmutability of one into the other disease. No doubt the conditions for the transmutation are not yet understood, but there are indications that the affirmative result obtained in a certain proportion of cases by the earlier observers may turn out to be an affair of conditions and circumstances of experiment. Particularly a note appended to Dr. Klein's tabular report (App. B. No. 2) is of interest in this respect, as showing how some unascertained difference in conditions of experiment have yielded in his hands very different results from those of the French Commission of 1865, when a certain kind of protective influence exerted upon the calf by inoculation of it with human small-pox was under investigation.

(*c.*) The chemical nature of the materials of disease, and the influences of disease on the normal chemistry of the body, form the subject of another class of investigations carried on under the auspices of the Board. This most recondite branch of pathological knowledge is studied by Dr. Thudichum, and it has needed to be prefaced by many profound researches into the chemistry of the body in health.

Papers by Dr. Thudichum on the chemical constitution of the brain and on the results of further researches into the chemistry of the organoplastic substances will be found in the Appendix B. No. 3 to this Report. During the year 1879 Dr. Thudichum made progress in his study of the nitrogenous principles of the brain, in continuation of his former researches into the chemistry of brain-compounds. It was foreseen (Medical Officer's Report, New Series, No. VIII.) that an inquiry of this great extent and extraordinary difficulty must needs occupy a considerable time; and I am glad now to be able to state that the completion of Dr. Thudichum's qualitative study of the various components of the healthy brain may be expected at no very distant period.

(*d.*) In the spring of 1877 the President directed that a portion of the Parliamentary grant which is made to the Board for purposes of scientific investigation should be appropriated to an inquiry to be undertaken by the Pathological Society of London into the nature, causes, and prevention of the infectious diseases known as pyæmia, septicæmia, and by other names. The report of the committee of the society charged with this study appears as Appendix B. No. 4, of the present volume. It considers the history of the so-called septic diseases during the ten years 1869–79 in the surgical practice of the chief London hospitals, and examines their incidences and their clinical phenomena. Passing on to microscopic examination of the blood and organs, the report records, among a variety of other morbid appearances, a very general presence of micrococci and bacteria, and considers the varieties of form of these organisms, and their distribution in one and another tissue or organ of the body. The relation between the several clinical manifestations of septic disease on the one hand and the observed particulate material on the other has not been made out; and the mechanism of the

morbid phenomena, as well as the meaning of the differences among them, must for the present remain unexplained. Not until much further study has been given, in a number of directions now more clearly seen, can the natural history of pyæmia and its allied diseases be understood. The committee refrain from suggesting any special measures for prevention, and their silence on this point will be seen to strengthen the demand for perfection in those common measures of sanitary care that are able, we know, to give a comparative security against these scourges of our hospitals.

I have the honour to be,
Sir,
Your obedient Servant,
GEORGE BUCHANAN.

August, 1880.

APPENDICES.

APPENDIX.

APPENDIX A.

No. 1.

DIGEST of the VACCINATION OFFICERS' RETURNS, with regard to CHILDREN whose BIRTHS were registered in the Year 1877.

The following return, which is the sixth annual return under the Vaccination Act, 1871, shows the continued successful working of that Act. Of 887,947* births returned to the Board by the several Vaccination Officers in England and Wales as registered during the year 1877, the number which, at the time the return was made, had been registered as successfully vaccinated was 766,824 (being 86·3 per cent. of the whole), and the number registered as having died before they could be vaccinated was 79,497 (or 8·9 per cent. of the whole). Of the remaining 41,626 children 926 (or 0·1 per cent. of the whole) had been registered as insusceptible of vaccination†; 118, or (·013 per cent.) as having contracted small-pox before they could be vaccinated; 6,681 (or 0·75 per cent.) as having their vaccination postponed by medical certificate; leaving 33,901 (or 3·8 per cent.) as "removed," "not to be traced," or otherwise unaccounted for. If from the 887,947 births returned by these officers deduction of the deaths without vaccination be first made, it appears that of the surviving 808,450 children, there were registered at the time of the return 94·85 per cent. as successfully vaccinated; 0·13 per cent. as either insusceptible of vaccination, or as having had small-pox; and 0·82 per cent. as under medical certificate of postponement; leaving 4·19 per cent. as at that time still unaccounted for as regards vaccination.

Of the 33,901 cases which this last per-centage represents, the very large majority were children who could not be found by the Vaccination Officers, usually because of the removal of the parents from the place of birth. In many districts the amount of removals without vaccination was unduly large, on account of delay on the part of the Vaccination Officer in instituting the inquiries which the Board's "Instructions to Vaccination Officers" direct. The remaining cases in the last column (with the exception of a certain number in which vaccination had really been performed successfully, but which could not be registered as vaccinated on account of the legal certificate of vaccination not having been received by the Vaccination Officer) were cases which at the time of the return were still unvaccinated in the union or district of birth. Local imperfections in administrative machinery were the main causes of this default, where it occurred. The number of cases in which non-vaccination was due to direct refusal on the part of parents to comply with the law when required to do so, constitutes, as usual, only a very small proportion of the cases entered in this column.

* This number does not include 95 births registered in the late Presteigne Union.

† The cases included under this heading are children, who being certified as having been three times unsuccessfully vaccinated, are no longer subject to the compulsory provisions of the law.

App. A. No. 1.
Digest of Vaccination Returns.

The results are still less complete in the metropolis than in the rest of the kingdom, and there is a slight falling off in the metropolitan returns for 1877 as compared with those for the previous year, the per-centage of cases unaccounted for having risen from 6·5 per cent. in 1876 to 7·1 per cent. in 1877. The per-centage in the rest of the kingdom, which was 4·0 in 1876, was 4·1 in 1877. Of the registered births of the six years 1872–7 the proportion not finally accounted for in regard to vaccination in each year respectively has been in the metropolis 8·8, 8·7, 8·8, 9·3, 6·5 and 7·1 per cent., while in the rest of the kingdom it has been only 4·5, 4·2, 4·1, 3·8, 4·0 and 4·1 per cent. In 1877 the proportion of cases unaccounted for, after deduction of the postponed cases, in the metropolis and in the rest of the kingdom, was 6·2 and 3·4 per cent. of births respectively.

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Smallpox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.	Percentage of Children not finally accounted for, including Cases postponed.
ENGLAND AND WALES.	887,947	766,324	926	118	79,497	6,681	33,901	4·5
Do. excluding Metropolitan Unions.	759,912	659,354	532	57	68,593	5,372	26,004	4·1
METROPOLITAN UNIONS.	128,035	107,470	394	61	10,904	1,309	*7,897	7·1
COUNTIES.								
Bedford	5,090	4,530	4	—	461	26	49	1·4
Berks	7,530	6,718	8	—	536	92	185	3·6
Bucks	5,130	4,532	7	—	480	33	78	2·1
Cambridge	6,288	5,570	2	—	536	67	113	2·8
Chester	20,563	18,265	13	12	1,787	88	398	2·3
Cornwall	9,962	8,854	3	—	1,018	100	187	2·8
Cumberland	8,754	7,697	2	—	802	62	191	2·9
Derby	14,208	12,404	10	—	1,337	54	398	3·1
Devon	18,218	16,242	16	—	1,460	162	338	2·7
Dorset	5,343	4,786	1	—	394	47	115	3·0
Durham	36,209	30,638	16	4	3,635	239	1,667	5·2
Essex	16,926	15,255	16	5	1,248	96	306	2·3
Gloucester	16,634	14,508	7	1	1,423	142	553	4·1
Hereford	3,389	2,976	2	—	255	29	127	4·6
Herts	7,276	6,622	2	2	513	32	105	1·8
Hunts	1,706	1,559	1	—	128	11	9	1·1
Kent (extra-metropolitan).	22,148	19,736	22	4	1,784	146	456	2·7
Lancaster	128,887	112,087	87	22	11,936	576	4,179	3·6
Leicester	11,854	9,597	8	—	1,378	66	805	7·3
Lincoln	14,887	12,382	12	—	1,488	221	784	6·7
Middlesex (extra-metropolitan).	9,573	8,331	21	2	750	37	431	4·8
Monmouth	9,061	8,042	4	—	702	26	197	2·4
Norfolk	13,319	11,666	10	1	1,322	89	231	2·4
Northampton	9,302	8,067	8	—	927	81	279	3·8
Northumberland	16,314	14,192	11	—	1,497	198	416	3·7
Nottingham	15,445	13,051	15	—	1,624	75	680	4·8
Oxford	5,750	5,023	4	—	474	28	221	4·3
Rutland	812	712	—	—	62	2	36	4·6
Salop	8,353	7,428	8	—	677	58	182	2·8
Somerset	15,121	13,375	7	—	1,241	146	352	3·2
Southampton	17,281	15,431	18	1	1,233	185	413	3·5
Stafford	35,884	29,449	13	1	3,141	94	1,186	3·7
Suffolk	11,359	10,106	9	—	895	84	265	3·0
Surrey (extra metropolitan).	13,084	11,579	24	1	910	121	449	4·3
Sussex	13,927	12,157	22	—	1,094	216	438	4·6
Warwick	26,848	22,537	25	—	2,327	96	1,363	5·5
Westmoreland	1,928	1,751	2	—	154	5	16	1·0
Wilts	7,682	6,985	3	—	513	91	90	2·3
Worcester	19,427	16,652	16	—	1,852	181	726	4·6
York, East Riding	12,603	10,548	14	—	1,244	96	701	6·3
York, North Riding	12,234	10,612	10	—	1,132	119	361	3·9
York, West Riding	80,134	66,502	31	1	7,983	620	4,997	7·0
WALES.								
Anglesey	994	877	1	—	90	3	23	2·6
Brecknock	1,885	1,602	1	—	189	8	55	3·3
Cardigan	2,310	1,992	—	—	155	18	45	2·8
Carmarthen	4,337	3,947	3	—	350	6	31	0·8
Carnarvon	3,851	3,379	4	—	395	37	36	1·8
Denbigh	2,827	2,433	1	—	299	30	64	3·3
Flint	2,911	2,587	2	—	253	19	50	2·3
Glamorgan	19,197	16,936	6	—	1,631	171	453	3·2
Merioneth	2,297	1,985	—	—	244	26	32	2·5
Montgomery	2,382	2,039	—	—	228	32	83	4·8
Pembroke	2,539	2,177	—	—	245	10	37	4·6
Radnor	514	446	—	—	51	5	12	3·3

UNIONS.

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.	Percentage of children not finally accounted for, including Cases postponed.
METROPOLITAN UNIONS.								
Bethnal Green	5,211	4,337	4	7	537	12	314	6·2
Camberwell	5,552	4,625	34	2	483	39	369	7·3
Chelsea	2,753	2,350	12	—	243	6	142	5·4
Fulham	3,427	2,979	7	—	284	19	138	4·5
George, St., Hanover Square	4,111	3,311	64	—	371	141	224	8·9
George St., in the East	1,838	1,382	—	—	175	18	263	15·3
Giles St. and George St.	1,670	1,325	2	1	144	16	182	11·8
Greenwich	4,301	3,724	15	3	334	9	216	5·2
Hackney	5,559	4,836	17	6	454	29	217	4·4
Hampstead	1,009	824	8	—	74	43	60	10·2
Holborn	5,836	4,935	15	—	540	18	328	5·9
Islington	9,383	7,719	7	2	711	307	637	10·0
Kensington	4,648	4,021	20	4	361	4	238	5·2
Lambeth	8,887	7,362	37	4	802	30	652	7·6
Lewisham	2,007	1,809	5	—	151	9	33	2·1
London, City	1,393	1,181	4	1	122	13	72	6·1
Marylebone	4,989	3,936	11	2	553	42	445	9·7
Mile End Old Town	4,149	3,411	18	7	385	20	308	7·9
Olave, St.	5,469	4,828	12	—	405	41	183	4·1
Paddington	2,907	2,416	10	—	222	67	192	8·9
Pancras, St.	8,092	6,913	18	5	569	21	566	7·2
Poplar	5,883	5,111	26	8	487	16	185	3·4
Saviour, St.	7,574	6,269	5	—	676	105	499	7 9
Shoreditch	5,135	3,938	2	—	451	108	636	14·5
Stepney	2,159	1,792	11	6	224	11	115	5·8
Strand	979	799	1	—	95	7	77	8·5
Wandsworth and Clapham	6,156	5,410	17	2	498	35	194	3·7
Westminster	1,390	1,222	2	—	103	8	55	4·5
Whitechapel	2,785	2,247	5	1	266	71	195	9·5
Woolwich	2,833	2,438	5	—	184	44	162	7·3
BEDFORDSHIRE.								
Ampthill	561	496	2	—	61	1	1	
Bedford	1,302	1,186	—	—	109	3	4	
Biggleswade	934	848	—	—	76	1	9	
Leighton Buzzard	664	595	2	—	54	—	13	
Luton	1,302	1,103	—	—	157	21	21	
Woburn	327	302	—	—	24	—	1	
BERKS.								
Abingdon	689	630	—	—	49	2	8	
Bradfield	538	498	—	—	36	3	1	
Cookham	500	439	—	—	29	9	23	
Easthampstead	326	300	3	—	15	3	5	
Faringdon	434	387	1	—	35	2	9	
Hungerford	532	478	1	—	48	1	4	
Newbury	594	522	2	—	42	20	8	
Reading	1,465	1,301	—	—	121	22	21	
Wallingford	424	381	—	—	29	2	12	
Wantage	526	479	1	—	39	2	5	
Windsor	920	796	—	—	55	4	65	
Wokingham	591	507	—	—	38	22	24	

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
BUCKS.							
Amersham	603	538	2	—	55	1	7
Aylesbury	811	702	—	—	87	7	15
Buckingham	409	360	2	—	34	2	11
Eton	752	687	1	—	44	6	14
Newport Pagnell	835	747	2	—	79	3	4
Winslow	242	208	—	—	25	2	7
Wycombe	1,478	1,290	—	—	156	12	20
CAMBRIDGE.							
Cambridge	1,059	942	1	—	87	15	14
Caxton and Arrington	356	321	—	—	25	—	—
Chesterton	970	876	—	—	61	11	22
Ely	755	633	—	—	89	8	25
Linton	430	381	—	—	25	11	13
Newmarket	884	799	—	—	74	5	6
North Witchford	519	472	—	—	36	3	8
Whittlesey	210	190	—	—	15	2	3
Wisbeach	1,105	946	1	—	124	12	22
CHESHIRE.							
Altrincham	1,788	1,641	3	1	106	6	31
Birkenhead	3,557	3,156	3	10	298	4	86
Tarvin	362	338	—	—	16	1	7
Chester	1,551	1,405	—	—	139	2	5
Congleton	1,317	1,174	—	—	127	5	11
Macclesfield	2,109	1,891	—	—	175	22	21
Nantwich	2,097	1,777	—	—	195	19	106
Northwich	1,668	1,497	—	—	156	4	11
Runcorn	1,274	1,154	1	—	103	2	14
Stockport	4,016	3,476	6	1	419	16	98
Wirrall	824	756	—	—	53	7	8
CORNWALL.							
Austell, St.	1,035	921	2	—	92	11	9
Bodmin	566	486	—	—	56	1	23
Camelford	260	235	—	—	22	2	1
Columb, St., Major	529	474	—	—	52	1	2
Falmouth	712	632	—	—	56	10	14
Germans, St.	502	465	—	—	34	1	2
Helston	718	608	1	—	83	10	16
Launceston	522	470	—	—	44	4	4
Liskeard	889	798	—	—	65	10	16
Penzance	1,504	1,282	—	—	176	18	28
Redruth	1,469	1,233	—	—	187	14	35
Stratton	228	199	—	—	20	4	5
Truro	1,028	851	—	—	131	14	32
CUMBERLAND.							
Alston with Garrigill	161	146	—	—	11	2	2
Bootle	463	412	—	—	37	1	13
Brampton	275	243	—	—	26	4	2
Carlisle	1,751	1,564	—	—	158	4	25
Cockermouth	2,030	1,774	—	—	200	19	37
Longtown	229	204	1	—	18	3	3
Penrith	695	644	—	—	46	2	3
Whitehaven	2,444	2,081	1	—	245	21	96
Wigton	706	629	—	—	61	6	10

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Digest of Vaccination Returns.

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
DERBY.							
Ashbourne	638	567	—	—	44	3	24
Bakewell	959	841	—	—	90	1	27
Belper	2,277	2,054	1	—	183	3	36
Chapel-en-le-Frith	607	536	—	—	44	10	17
Chesterfield	4,144	3,630	6	—	385	24	99
Derby	2,903	2,491	1	—	328	—	83
Glossop	714	598	1	—	86	3	26
Hayfield	433	358	—	—	52	7	16
Shardlow	1,528	1,329	1	—	125	3	70
DEVON.							
Axminster	587	517	—	—	53	2	15
Barnstaple	1,077	957	—	—	96	13	11
Bideford	539	504	—	—	29	3	3
Crediton	547	499	—	—	30	8	10
East Stonehouse	565	500	—	—	58	2	5
Exeter	1,095	908	1	—	133	21	32
Holsworthy	279	264	—	—	13	1	1
Honiton	662	587	—	—	60	3	12
Kingsbridge	607	559	1	—	41	—	6
Newton Abbot	2,049	1,853	4	—	136	21	35
Okehampton	573	519	—	—	42	7	5
Plymouth	2,239	1,877	4	—	263	26	69
Plympton St. Mary	697	616	1	—	53	17	10
South Molton	502	456	—	—	32	4	10
Stoke Damerel	1,470	1,303	—	—	114	10	43
Tavistock	849	788		—	50	1	10
Thomas, St.	1,434	1,302	—	—	96	10	26
Tiverton	797	704	3	—	62	5	23
Torrington	452	408	1	—	34	3	6
Totnes	1,198	1,121	1	—	65	5	6
DORSET.							
Beaminster	359	320	1	—	26	5	7
Blandford	378	336	—	—	28	4	10
Bridport	504	456	—	—	37	3	8
Cerne	200	176	—	—	20	4	—
Dorchester	498	461	—	—	31	1	5
Poole	604	543	—	—	34	—	27
Shaftesbury	385	331	—	—	31	13	10
Sherborne	344	320	—	—	23	—	1
Sturminster	309	267	—	—	39	1	2
Wareham and Purbeck	396	456	—	—	30	6	4
Weymouth	794	692	—	—	63	8	31
Wimborne and Cranborne	472	428	—	—	32	2	10
DURHAM.							
Auckland	3,806	3,228	3	2	394	7	172
Chester-le-Street	1,856	1,622	1	—	205	4	24
Darlington	1,924	1,644	2	1	168	6	103
Durham	2,823	2,377	5	1	327	21	92
Easington	1,878	1,543	—	—	169	16	150
Gateshead	4,454	3,605	2	—	439	16	392
Hartlepool	1,966	1,740	—	—	183	19	24
Houghton-le-Spring	1,491	1,295	—	—	164	1	31
Lanchester	2,419	2,132	1	—	215	11	60

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
DURHAM—*cont.*							
Sedgefield	908	776	—	—	94	8	30
South Shields	3,941	3,247	2	—	393	61	238
Stockton	2,000	1,671	—	—	223	—	106
Sunderland	5,299	4,462	—	—	529	59	249
Teesdale	691	525	—	—	49	12	5
Weardale	753	661	—	—	83	8	1
ESSEX.							
Billericay	475	415	—	—	38	4	23
Braintree	531	470	1	—	54	—	6
Chelmsford	1,081	985	—	—	83	4	9
Colchester	912	803	1	—	72	13	23
Dunmow	558	516	—	—	37	—	5
Epping	644	571	1	—	53	5	14
Halstead	513	462	—	—	41	5	5
Lexden and Winstree	782	716	—	—	61	1	4
Maldon	772	700	1	—	51	3	17
Ongar	321	288	1	—	22	2	8
Orsett	496	453	1	—	33	2	7
Rochford	760	686	—	—	42	8	24
Romford	1,232	1,114	1	—	90	1	26
Saffron Walden	589	530	2	—	43	8	6
Tendring	932	856	—	2	60	5	9
West Ham	5,883	5,287	6	3	442	30	115
Witham	445	403	1	—	31	5	5
GLOUCESTER.							
Barton Regis	5,482	4,841	3	1	455	6	176
Bristol	2,085	1,749	1	—	205	37	93
Cheltenham	1,385	1,064	—	—	152	37	132
Chipping Sodbury	521	462	—	—	47	4	8
Cirencester	622	555	—	—	48	6	13
Dursley	336	307	—	—	23	3	3
Gloucester	1,484	1,282	1	—	144	8	49
Newent	336	316	—	—	15	2	3
Northleach	305	273	—	—	25	4	3
Stow-on-the-Wold	277	238	—	—	20	6	13
Stroud	1,271	1,135	—	—	115	8	13
Tetbury	190	163	1	—	23	1	2
Tewkesbury	390	351	—	—	33	1	5
Thornbury	567	516	1	—	39	7	4
Westbury-on-Severn	872	779	—	—	51	11	31
Wheatenhurst	220	208	—	—	11	—	1
Winchcomb	291	269	—	—	17	1	4
HEREFORD.							
Bromyard	322	287	—	—	25	2	8
Dore	244	223	—	—	15	4	2
Hereford	989	899	—	—	65	11	14
Kington	343	310	—	—	24	—	9
Ledbury	319	278	—	—	31	1	9
Leominster	411	367	2	—	30	—	12
Ross	528	406	—	—	52	10	60
Weobley	233	206	—	—	13	1	13

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—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
HERTS.							
Albans, St.	766	702	—	—	56	1	7
Barnet	875	797	—	—	52	1	25
Berkhampstead	509	448	—	—	49	6	6
Bishop's Stortford	711	633	—	1	64	5	8
Buntingford	177	163	—	—	9	4	1
Hatfield	204	180	—	—	18	1	5
Hemel Hempstead	460	420	—	—	32	1	7
Hertford	478	444	—	—	17	4	13
Hitchin	918	841	—	—	70	—	7
Royston	651	591	—	—	56	4	—
Ware	568	517	—	—	33	3	15
Watford	894	825	2	1	54	2	10
Welwyn	65	61	—	—	3	—	1
HUNTINGDON.							
Huntingdon	607	549	1	—	49	3	5
Ives, St.	572	521	—	—	43	4	4
Neots, St.	529	489	—	—	36	4	—
KENT (extra metropolitan).							
Ashford, East	444	411	—	—	26	1	6
Ashford, West	586	518	—	—	45	4	19
Blean	633	581	—	—	45	—	7
Bridge	396	360	—	—	29	5	2
Bromley	1,429	1,209	2	—	145	15	58
Canterbury	559	482	1	—	55	—	21
Cranbrook	422	388	—	—	23	8	3
Dartford	1,532	1,364	—	—	126	12	30
Dover	1,105	972	3	—	98	9	23
Eastry	749	647	1	—	68	6	32
Elham	1,044	925	—	—	81	18	20
Faversham	847	752	1	—	81	3	10
Gravesend and Milton	706	640	1	—	55	2	8
Hollingbourn	481	436	—	—	33	1	11
Hoo	112	97	—	—	12	—	3
Maidstone	1,490	1,322	2	—	135	5	26
Malling	899	817	—	—	58	7	17
Medway	2,103	1,880	3	2	168	16	34
Milton	847	768	2	—	68	2	7
North Aylesford	892	805	1	2	70	—	14
Romney Marsh	195	184	—	—	11	—	—
Sevenoaks	793	725	1	—	44	4	19
Sheppey	590	528	—	—	40	4	18
Tenterden	332	297	—	—	28	1	6
Thanet, Isle of	1,403	1,242	2	—	123	15	21
Tonbridge	1,559	1,386	2	—	117	13	41
LANCASHIRE.							
Ashton-under-Lyne	5,732	5,023	6	1	538	39	125
Barrow-in-Furness	1,895	1,671	—	—	133	17	74
Barton-upon-Irwell	2,360	2,066	—	—	179	1	114
Blackburn	6,480	5,842	3	—	592	13	30
Bolton	7,283	6,589	2	—	561	2	129
Burnley	4,142	3,604	3	—	438	19	78
Bury	4,722	4,161	3	—	421	18	119

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
LANCASHIRE—*cont.*							
Chorley	1,797	1,555	—	—	186	20	36
Chorlton	9,364	7,912	6	—	882	4	560
Clitheroe	736	663	1	—	61	—	11
Fylde, The	1,298	1,136	—	—	110	2	50
Garstang	351	322	—	—	18	2	9
Haslingden	3,373	3,066	—	—	288	4	15
Lancaster	1,256	1,089	—	1	131	11	24
Leigh	2,071	1,831	2	—	184	4	50
Liverpool	7,664	6,515	8	4	842	7	288
Lunesdale	213	194	—	—	17	2	—
Manchester	6,546	5,501	3	3	695	54	290
Oldham	6,084	5,104	5	4	625	36	310
Ormskirk	2,663	2,367	3	—	213	8	72
Prescot	4,780	4,042	—	—	481	53	204
Preston	4,706	3,983	1	3	542	7	170
Prestwich	3,991	3,499	2	—	314	19	157
Rochdale	4,288	3,723	3	—	395	29	138
Salford	7,112	6,151	8	1	697	30	225
Todmorden	1,170	1,043	—	—	109	3	15
Toxteth Park	4,005	3,340	5	2	333	3	322
Ulverstone	1,592	1,405	1	2	119	27	38
Warrington	2,670	2,320	2	—	208	32	108
West Derby	12,495	10,941	19	1	1,113	86	335
Wigan	6,048	5,429	1	—	511	24	83
LEICESTER.							
Ashby-de-la-Zouch	1,401	1,189	—	—	146	6	60
Barrow-on-Soar	973	771	—	—	101	8	93
Billesdon	278	239	—	—	27	1	11
Blaby	674	584	—	—	71	6	13
Hinckley	671	591	—	—	79	—	1
Leicester	4,749	3,509	7	—	647	37	549
Loughborough	1,012	876	—	—	123	3	10
Lutterworth	353	325	—	—	26	—	2
Market Bosworth	539	484	1	—	48	1	5
Market Harborough	492	421	—	—	54	2	15
Melton Mowbray	712	608	—	—	56	2	46
LINCOLNSHIRE.							
Boston	1,245	1,033	—	—	137	6	69
Bourn	612	523	—	—	56	13	20
Caistor	2,246	1,766	6	—	252	18	204
Gainsborough	932	744	1	—	89	18	80
Glanford Brigg	1,373	1,184	1	—	131	15	42
Grantham	1,052	915	1	—	104	11	21
Holbeach	659	591	—	—	45	8	15
Horncastle	666	601	—	—	49	—	16
Lincoln	2,117	1,574	1	—	237	92	213
Louth	1,047	897	2	—	107	18	23
Sleaford	795	718	—	—	71	—	6
Spalding	751	660	—	—	72	4	15
Spilsby	868	709	—	—	96	15	48
Stamford	524	467	—	—	42	3	12

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—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Smallpox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
MIDDLESEX (extra metropolitan).							
Brentford	2,830	2,285	6	—	250	13	276
Edmonton	3,631	3,261	10	1	268	11	80
Hendon	1,565	1,412	3	1	105	4	40
Staines	718	621	1	—	67	7	22
Uxbridge	828	752	1	—	60	2	13
MONMOUTHSHIRE.							
Abergavenny	889	779	1	—	91	7	11
Bedwellty	2,312	2,003	—	—	210	—	99
Chepstow	615	558	—	—	46	2	9
Monmouth	1,093	977	1	—	94	3	18
Newport	2,714	2,434	2	—	228	12	38
Pontypool	1,438	1,291	—	—	123	2	22
NORFOLK.							
Aylsham	515	446	—	—	52	10	7
Blofield	325	295	—	—	28	—	2
Depwade	677	560	4	—	78	7	28
Docking	515	447	1	—	60	—	7
Downham	587	524	—	—	53	2	8
Erpingham	581	519	—	—	45	7	10
Faith, St.	324	294	—	—	26	1	3
Flegg, East and West	356	310	—	—	33	1	12
Forehoe	361	322	—	—	30	—	9
Freebridge Lynn	408	355	—	—	35	12	6
Guiltcross	289	248	—	—	32	2	7
Henstead	315	283	1	—	29	—	2
King's Lynn	518	454	—	—	48	5	11
Loddon and Clavering	390	352	—	—	28	3	7
Mitford and Launditch	861	777	—	—	74	3	7
Norwich	2,819	2,472	3	1	306	1	36
Swaffham	408	358	—	—	31	12	7
Thetford	552	496	—	—	43	—	13
Smallburgh	423	372	—	—	43	—	8
Walsingham	559	488	—	—	52	1	18
Wayland	334	281	1	—	41	7	4
Yarmouth, Great	1,202	1,013	—	—	155	15	19
NORTHAMPTONSHIRE.							
Brackley	402	330	—	—	46	1	25
Brixworth	394	364	—	—	24	1	5
Daventry	549	488	—	—	43	4	14
Hardingstone	329	294	—	—	28	2	5
Kettering	881	776	—	—	84	2	19
Northampton	2,273	1,869	1	—	274	18	111
Oundle	457	412	3	—	34	—	8
Peterborough	1,461	1,318	1	—	126	—	16
Potterspury	359	314	—	—	34	4	7
Thrapston	553	502	3	—	41	—	7
Towcester	409	353	—	—	41	2	13
Wellingborough	1,295	1,047	—	—	152	47	49

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Smallpox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
NORTHUMBERLAND.							
Alnwick	632	558	—	—	49	10	15
Belford	140	132	—	—	8	—	—
Bellingham	195	167	1	—	18	—	9
Berwick-on-Tweed	690	615	—	—	59	11	5
Castle Ward	722	642	—	—	69	—	11
Glendale	302	275	—	—	20	1	6
Haltwhistle	253	229	—	—	21	2	1
Hexham	1,190	1,031	—	—	93	8	58
Morpeth	1,671	1,481	4	—	149	9	28
Newcastle-on-Tyne	5,789	4,991	3	—	556	58	181
Rothbury	181	166	—	—	12	1	2
Tynemouth	4,549	3,905	3	—	443	98	100
NOTTINGHAMSHIRE.							
Basford	4,747	4,059	7	—	528	16	137
Bingham	501	439	—	—	41	2	19
East Retford	735	656	—	—	59	2	18
Mansfield	1,768	1,502	1	—	186	24	55
Newark	997	891	1	—	81	3	21
Nottingham	3,498	2,770	4	—	400	22	302
Radford	1,500	1,283	—	—	180	4	83
Southwell	635	568	1	—	49	—	17
Worksop	1,064	983	1	—	100	2	28
OXFORDSHIRE.							
Banbury	969	745	2	—	106	13	103
Bicester	465	415	—	—	34	4	12
Chipping Norton	664	586	—	—	62	3	13
Headington	821	723	1	—	61	—	36
Henley	624	566	—	—	42	4	12
Oxford	665	585	—	—	53	2	25
Thame	430	402	—	—	23	—	5
Witney	701	645	—	—	52	—	4
Woodstock	411	356	1	—	41	2	11
RUTLAND.							
Oakham	320	287	—	—	28	2	3
Uppingham	492	425	—	—	34	—	33
SALOP.							
Atcham	1,356	1,194	—	—	127	13	22
Bridgnorth	448	393	—	—	30	2	18
Church Stretton	169	151	—	—	14	1	3
Cleobury Mortimer	242	212	—	—	20	4	6
Clun	312	264	—	—	19	5	24
Drayton	456	406	1	—	41	—	8
Ellesmere	365	338	—	—	24	1	2
Ludlow	571	487	3	—	47	11	23
Madeley	1,080	953	2	—	83	9	33
Newport	483	432	1	—	37	7	6
Oswestry	859	761	1	—	84	2	11
Shifnal	412	378	—	—	25	—	9
Wellington	997	909	—	—	79	2	7
Wem	252	237	—	—	12	1	2
Whitchurch	356	313	—	—	35	—	8

APP. A. No. 1.

Digest of Vaccination Returns.

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
SOMERSET.							
Axbridge	1,147	1,032	1	—	91	8	15
Bath	1,818	1,491	—	—	195	16	116
Bedminster	2,463	2,106	2	—	261	53	41
Bridgwater	1,058	936	—	—	75	14	33
Chard	862	776	—	—	69	7	10
Clutton	794	739	—	—	50	2	3
Dulverton	159	151	—	—	6	1	1
Frome	791	705	—	—	66	2	18
Keynsham	849	769	—	—	71	—	9
Langport	475	415	1	—	30	6	23
Shepton Mallet	440	400	1	—	30	7	2
Taunton	964	867	2	—	69	9	17
Wellington	546	510	—	—	28	2	6
Wells	651	579	—	—	54	7	11
Williton	597	556	—	—	26	6	9
Wincanton	640	578	—	—	46	1	15
Yeovil	867	765	—	—	74	5	23
SOUTHAMPTON.							
Alresford	239	223	1	—	11	2	2
Alton	492	429	1	1	37	3	21
Alverstoke	726	668	—	—	54	2	2
Andover	478	437	—	—	30	3	8
Basingstoke	568	498	—	—	35	13	22
Catherington	68	54	—	—	10	—	4
Christchurch	749	670	1	—	39	19	20
Droxford	363	335	—	—	19	2	7
Fareham	531	482	—	—	29	4	16
Fordingbridge	190	182	—	—	8	—	—
Hartley Wintney	567	510	2	—	31	6	18
Havant	257	239	1	—	13	1	3
Hursley	80	77	—	—	3	—	—
Kingsclere	259	234	—	—	21	1	3
Lymington	350	320	—	—	14	11	5
New Forest	384	348	—	—	23	4	9
Petersfield	312	281	—	—	25	1	5
Portsea Island	4,111	3,697	7	—	329	16	62
Ringwood	151	142	—	—	7	1	1
Romsey	322	291	—	—	18	1	12
Southampton	1,697	1,488	—	—	134	12	63
South Stoneham	1,195	1,019	—	—	97	24	55
Stockbridge	190	177	1	—	11	—	1
Whitchurch	173	138	1	—	20	3	11
Wight, Isle of	2,036	1,788	3	—	148	47	50
Winchester, New	793	704	—	—	67	9	13
STAFFORDSHIRE.							
Wolstanton and Burslem	3,305	2,918	—	—	361	5	21
Burton-on-Trent	2,666	2,170	—	—	237	19	240
Cannock	1,519	1,290	—	—	162	10	57
Cheadle	787	692	—	—	72	4	19
Leek	1,161	1,022	—	1	100	5	33
Lichfield	1,322	1,184	1	—	93	8	36
Newcastle-under-Lyme	1,354	1,128	—	—	107	—	119
Seisdon	529	480	—	—	39	—	10
Stafford	1,043	880	2	—	100	3	58
Stoke-upon-Trent	4,349	3,760	1	—	435	9	144

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
STAFFORDSHIRE—*cont.*							
Stone	904	800	1	—	88	—	15
Tamworth	701	630	—	—	48	4	19
Uttoxeter	405	371	1	—	22	—	11
Walsall	3,600	3,026	2	—	389	13	170
West Bromwich	5,057	4,462	3	—	454	7	131
Wolverhampton	5,182	4,636	2	—	434	7	103
SUFFOLK.							
Blything	771	677	—	—	65	9	20
Bosmere and Claydon	517	448	1	—	47	3	18
Bury St. Edmunds	490	426	—	—	53	3	8
Cosford	488	431	3	—	45	—	9
Hartismere	531	485	—	—	36	1	9
Hoxne	390	346	—	—	31	3	10
Ipswich	1,633	1,425	2	—	147	26	33
Mildenhall	283	261	—	—	16	—	6
Mutford and Lothingland	1,314	1,142	2	—	104	6	60
Plomesgate	636	585	—	—	37	5	9
Risbridge	591	545	—	—	42	—	4
Samford	383	342	1	—	30	—	7
Stow	694	624	—	—	57	5	8
Sudbury	999	903	—	—	76	4	16
Thingoe	511	459	—	—	37	2	13
Wangford	462	394	—	—	35	7	26
Woodbridge	669	613	—	—	37	10	9
SURREY (extra metropolitan).							
Chertsey	800	698	3	—	46	19	34
Croydon	3,255	2,789	6	—	257	33	170
Dorking	468	425	—	—	36	—	7
Epsom	1,012	884	—	—	69	23	36
Farnham	1,532	1,330	2	—	107	2	91
Godstone	403	371	—	—	22	3	7
Guildford	1,108	1,015	—	—	66	8	19
Hambledon	501	465	1	—	32	—	3
Kingston	2,213	1,964	3	1	157	30	58
Reigate	867	804	5	—	43	2	13
Richmond	925	834	4	—	75	1	11
SUSSEX.							
Battle	415	385	—	—	24	4	2
Brighton	2,807	2,480	8	—	269	18	37
Chailey	292	265	—	—	24	2	1
Chichester	242	203	1	—	14	17	7
Cuckfield	693	623	—	—	41	9	20
Eastbourne	684	582	4	—	55	19	24
East Grinstead	588	531	—	—	39	6	12
East Preston	681	600	—	—	61	3	17
Hailsham	452	400	—	—	36	5	11
Hastings	1,315	1,004	1	—	125	46	139
Horsham	644	563	2	—	53	11	15
Lewes	308	269	—	—	17	15	7

App. A. No. 1.

Digest of Vaccination Returns.

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Smallpox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
SUSSEX—*cont.*							
Midhurst	439	405	—	—	30	2	2
Newhaven	211	166	8	—	20	—	17
Petworth	295	282	—	—	8	2	3
Rye	359	324	2	—	23	—	10
Steyning	1,164	952	—	—	110	37	65
Thakeham	283	259	1	—	16	1	6
Ticehurst	540	479	—	—	38	5	18
Uckfield	661	608	—	—	42	4	7
Westbourne	227	207	—	—	13	—	7
West Firle	59	53	—	—	5	—	1
West Hampnett	568	517	—	—	31	10	10
WARWICKSHIRE.							
Alcester	590	525	—	—	42	16	7
Aston	8,480	7,176	1	—	735	15	503
Atherstone	491	436	1	—	44	2	8
Birmingham	10,026	8,365	18	—	931	21	691
Coventry	1,556	1,368	—	—	139	11	38
Foleshill	712	623	—	—	66	2	21
Meriden	290	269	—	—	19	1	1
Nuneaton	486	420	—	—	49	10	7
Rugby	770	677	1	—	76	1	15
Solihull	519	456	1	—	44	4	14
Southam	335	308	—	—	19	3	5
Stratford-on-Avon	715	640	—	—	37	1	37
Warwick	1,428	1,274	3	—	126	9	16
WESTMORELAND.							
East Ward	462	398	1	—	53	2	8
Kendal	1,247	1,152	1	—	83	3	8
West Ward	219	201	—	—	18	—	—
WILTSHIRE.							
Alderbury	746	687	—	—	49	3	7
Amesbury	226	206	—	—	15	—	5
Bradford	354	325	—	—	24	2	3
Calne	253	232	—	—	14	4	3
Chippenham	664	598	1	—	42	19	4
Cricklade and Wootton Bassett.	373	334	—	—	22	12	5
Devizes	596	546	—	—	38	6	6
Highworth and Swindon	1,286	1,153	—	—	98	20	15
Malmesbury	459	410	—	—	32	13	4
Marlborough	275	253	—	—	22	—	—
Melksham	573	525	—	—	38	4	6
Mere	240	228	—	—	12	—	—
Pewsey	357	322	—	—	23	—	12
Tisbury	252	219	—	—	21	4	8
Warminster	358	333	—	—	16	2	7
Westbury and Whorwelsdown.	374	351	2	—	19	1	1
Wilton	296	263	—	—	28	1	4

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
WORCESTERSHIRE.							
Bromsgrove	1,076	953	—	—	105	6	12
Droitwich	778	693	—	—	63	3	19
Dudley	6,424	5,400	3	—	702	28	291
Evesham	502	437	2	—	47	3	13
Kidderminster	1,334	1,225	—	—	93	5	11
King's Norton	2,865	2,327	8	—	256	110	164
Martley	449	413	—	—	23	2	11
Pershore	346	309	—	—	33	1	3
Shipston-on-Stour	548	486	—	—	46	3	13
Stourbridge	3,294	2,881	2	—	330	3	78
Tenbury	222	195	—	—	15	4	8
Upton-on-Severn	562	518	—	—	31	7	6
Worcester	1,027	815	1	—	108	6	97
YORKSHIRE.							
East Riding.							
Beverley	773	645	1	—	79	11	37
Bridlington	524	437	1	—	52	5	29
Driffield	715	612	—	—	79	9	15
Howden	408	361	—	—	37	2	8
Kingston-upon-Hull	3,002	2,584	6	—	315	8	89
Patrington	285	240	—	—	40	1	4
Pocklington	482	408	—	—	53	10	11
Sculcoates	3,645	3,083	6	—	363	20	173
Skirlaugh	333	303	—	—	23	3	4
York	2,436	1,875	—	—	203	27	331
YORKSHIRE.							
North Riding.							
Aysgarth	190	157	—	—	27	1	5
Bedale	249	221	—	—	25	1	2
Easingwold	289	261	—	—	27	—	1
Guisborough	2,009	1,713	—	—	183	11	102
Helmsley Blackmoor	190	176	—	—	13	—	1
Kirkby Moorside	159	136	—	—	17	1	5
Leyburn	250	222	—	—	26	1	1
Malton	753	632	1	—	67	12	41
Middlesbrough	3,691	3,229	5	—	340	18	99
Northallerton	379	339	—	—	29	5	6
Pickering	469	376	—	—	58	14	21
Reeth	161	144	—	—	12	4	1
Richmond	443	400	—	—	33	2	8
Scarborough	1,274	1,078	4	—	131	25	36
Stokesley	411	373	—	—	33	3	2
Thirsk	432	384	—	—	27	7	14
Whitby	885	771	—	—	84	14	16
YORKSHIRE.							
West Riding.							
Barnsley	3,373	2,846	1	—	310	27	189
Bramley	2,114	1,859	—	—	198	5	52
Bradford	6,794	5,529	2	1	724	10	528
Bierley, North	4,735	4,009	—	—	483	49	194
Dewsbury	5,815	4,048	3	—	733	86	945
Doncaster	1,763	1,507	4	—	209	4	39

App. A. No. 1.

Digest of Vaccination Returns.

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
YORKSHIRE.							
West Riding—*cont.*							
Ecclesall Bierlow	3,934	3,385	7	—	325	27	190
Goole	701	618	1	—	64	2	16
Halifax	5,861	5,074	1	—	547	82	157
Hemsworth	345	298	—	—	33	1	13
Holbeck	964	844	—	—	100	3	17
Huddersfield	5,226	4,707	1	—	461	16	41
Hunslet	2,463	2,109	—	—	266	4	84
Keighley	2,109	680	1	—	285	54	1,089
Knaresborough	592	484	3	—	74	6	25
Leeds	7,244	6,443	—	—	688	10	103
Ouseburn, Great	363	330	1	—	27	—	5
Pateley Bridge	275	254	—	—	20	—	1
Penistone	642	576	—	—	48	4	14
Pontefract	2,121	1,775	1	—	194	24	127
Ripon	504	441	—	—	46	11	6
Rotherham	3,125	2,698	—	—	247	12	168
Saddleworth	736	619	—	—	83	2	32
Sedbergh	131	112	—	—	15	2	2
Selby	527	468	—	—	51	4	4
Settle	484	434	—	—	37	4	9
Sheffield	7,488	6,221	4	—	732	38	493
Skipton	1,218	953	—	—	153	59	53
Tadcaster	815	697	—	—	77	3	38
Thorne	548	466	—	—	68	1	13
Wakefield	3,647	3,143	—	—	333	2	169
Wetherby	421	295	—	—	47	29	50
Wharfedale	1,617	1,336	1	—	172	27	81
Wortley	1,439	1,244	—	—	133	12	50
ANGLESEY.							
Anglesey	412	360	1	—	45	—	6
Holyhead	582	517	—	—	45	3	17
BRECKNOCK.							
Brecknock	525	472	—	—	48	2	3
Builth	253	227	1	—	22	1	2
Crickhowell	758	624	—	—	83	1	50
Hay	319	279	—	—	36	4	—
CARDIGAN.							
Aberaeron	352	313	—	—	26	1	12
Aberystwith	767	680	—	—	54	8	25
Cardigan	485	445	—	—	38	—	2
Lampeter	296	277	—	—	15	1	3
Tregaron	310	277	—	—	22	8	3
CARMARTHEN.							
Carmarthen	1,053	963	—	—	82	2	6
Llanelly	1,704	1,553	3	—	128	1	19
Llandilo Fawr	580	527	—	—	48	1	4
Llandovery	455	395	—	—	57	2	1
Newcastle-in-Emlyn	545	509	—	—	35	—	1

—	Births.	Successfully Vaccinated.	Insusceptible of Vaccination.	Had Small-pox.	Dead, Unvaccinated.	Vaccination postponed.	Remaining.
CARNARVON.							
Bangor and Beaumaris	1,146	1,032	1	—	104	5	4
Carnarvon	1,542	1,314	8	—	190	19	16
Conway	511	447	—	—	50	3	11
Pwllheli	652	586	—	—	51	10	5
DENBIGH.							
Llanrwst	404	342	—	—	47	11	4
Ruthin	380	347	—	—	33	—	—
Wrexham	2,043	1,744	1	—	219	19	60
FLINT.							
Asaph, St.	796	718	—	—	74	2	2
Hawarden	556	506	2	—	41	1	6
Holywell	1,559	1,363	—	—	138	16	42
GLAMORGAN.							
Bridgend and Cowbridge	1,398	1,284	—	—	98	4	12
Cardiff	3,504	3,036	—	—	303	68	97
Gower	407	360	—	—	36	6	5
Merthyr Tydfil	3,888	3,514	1	—	306	20	47
Neath	2,019	1,814	—	—	149	20	36
Pontardawe	853	770	—	—	67	6	10
Pontypridd	3,589	3,046	4	—	392	19	128
Swansea	3,539	3,112	1	—	280	28	118
MERIONETH.							
Bala	155	140	—	—	8	4	3
Corwen	589	507	—	—	63	5	14
Dolgelly	519	457	—	—	54	4	4
Festiniog	1,024	881	—	—	119	13	11
MONTGOMERY.							
Llanfyllin	560	487	—	—	66	3	4
Machynlleth	392	342	—	—	33	3	9
Forden	542	489	—	—	38	9	6
Newtown and Llanidloes	888	721	—	—	91	12	64
PEMBROKE.							
Haverfordwest	999	872	—	—	92	19	16
Narberth	599	508	—	—	55	23	13
Pembroke	941	797	—	—	98	38	8
RADNOR.							
*Knighton	305	260	—	—	36	3	6
Rhayader	209	186	—	—	15	2	6

* The Presteigne Union was dissolved in 1877, and added to the Knighton and Kington Unions. No return relating to children born in Presteigne during that year has been received. The number of births registered was 95.

APP. A. No. 2.

Inspection of Public Vaccination.

No. 2.

LIST (alphabetically arranged) of 348 UNIONS inspected during the Year 1879, with reference to the PROCEEDINGS under the Vaccination Acts, 1867 and 1871, and an ACCOUNT of the AWARDS made to the respective PUBLIC VACCINATORS out of Moneys voted by Parliament for that Purpose.

Union.	No. of Vaccination Districts in the Union.	No. of respective Vaccination Contractors recommended for Award.		Range of Awards in each Union.		Total Sum awarded in the Union.	Inspector.
		1st Class Award.	2nd Class Award.	Minimum.	Maximum.		
				£ s. d.	£ s. d.	£ s. d.	
Abergavenny	2	2	—	12 1 0	12 8 0	24 9 0	Dr. Airy.
Aberystwith	4	3	1	9 9 0	13 17 0	46 17 4	Do.
Alcester	5	2	—	5 14 0	13 1 0	18 15 0	Mr. Power.
Alderbury	5	3	—	4 5 0	25 16 0	40 6 0	Dr. Blaxall.
Amersham	6	3	—	4 10 0	18 14 0	29 12 0	„ Thorne.
Amesbury	2	1	—	—	—	5 18 0	„ Blaxall.
Anglesey	3	—	1	—	—	10 8 0	„ Parsons.
Asaph, St.	5	1	1	6 9 4	7 6 0	13 15 4	Do.
Ashford, East	7	6	—	2 0 0	7 7 0	33 16 0	„ Stevens.
Ashford, West	3	3	—	6 19 0	19 12 0	36 12 0	Do.
Ashton-under-Lyne	12	5	—	12 10 0	41 19 0	150 12 0	„ Beard.
Aston	7	6	1	1 19 4	112 0 0	379 9 4	Mr. Power.
Atcham	7	3	1	3 18 0	36 0 0	51 1 0	Dr. Blaxall.
Atherstone	2	2	—	14 3 0	21 19 0	36 2 0	Mr. Power.
Auckland	9	7	—	11 15 0	38 9 0	143 12 0	Do.
Aylesbury	7	7	—	2 11 0	10 11 0	49 19 0	Dr. Thorne.
Aysgarth	2	—	—	—	—	—	Mr. Power.
Bala	1	1	—	—	—	11 1 0	Dr. Parsons.
Banbury	7	6	—	1 7 0	13 5 0	43 13 0	„ Thorne.
Bangor & Beaumaris	5	—	—	—	—	—	„ Parsons.
Barnsley	5	1	—	—	—	16 16 0	„ Beard.
Barton-upon-Irwell	6	3	—	4 7 0	37 18 0	58 12 0	Do.
Battle	6	3	—	5 19 0	12 17 0	30 5 0	„ Stevens.
Beaminster	5	2	1	1 8 0	7 10 0	11 0 0	„ Blaxall.
Bedale	3	3	—	2 19 0	9 19 0	16 18 0	Mr. Power.
Bethnal Green	2	2	—	69 15 0	189 2 0	258 17 0	Dr. Stevens.
Bicester	7	4	—	0 13 0	11 4 0	21 1 0	„ Thorne.
Billericay	5	3	1	2 10 0	9 9 4	24 1 4	„ Airy.
Birmingham	1	1	—	—	—	265 11 0	„ Stevens.
Blandford	4	3	—	3 14 0	9 14 0	20 17 0	„ Blaxall.
Blean	4	2	1	3 0 0	12 1 0	19 8 0	„ Stevens.
Bolton	8	7	—	2 19 0	83 2 0	236 9 0	„ Beard.
Bootle	3	2	—	3 19 0	19 10 0	23 9 0	Mr. Power.
Brackley	5	4	—	3 9 0	4 15 0	16 13 0	Dr. Thorne.
Bradford (Wilts)	3	—	—	—	—	—	„ Blaxall.
Bradford (W. York)	4	3	—	45 14 0	59 13 0	155 19 0	„ Beard.
Braintree	4	4	—	4 12 0	18 1 0	43 13 0	„ Airy.
Bramley	5	2	—	20 12 0	20 15 0	41 7 0	„ Beard.
Brecknock	3	2	—	10 12 0	20 5 0	30 17 0	„ Airy.
Brentford	8	5	—	8 16 0	20 6 0	77 5 0	„ Thorne.
Bridge	5	5	—	3 13 0	7 16 0	29 13 0	„ Stevens.
Bridgend&Cowbridge	4	—	—	—	—	—	„ Airy.
Bridgnorth	3	3	—	3 6 0	15 16 0	27 8 0	„ Blaxall.
Bridport	4	1	1	5 14 0	10 10 8	16 4 8	Do.
Brighton	2	2	—	62 10 0	80 2 0	142 12 0	„ Stevens.
Bristol	1	1	—	—	—	51 17 0	Do.
Brixworth	6	5	—	1 2 0	5 12 0	17 11 0	„ Thorne.
Bromley	5	4	—	2 14 0	19 19 0	44 18 0	„ Stevens.
Buckingham	4	2	—	6 11 0	8 19 0	15 10 0	„ Thorne.
Builth	1	1	—	—	—	20 8 0	„ Airy.
Burnley	7	6	—	1 6 0	32 2 0	82 7 0	„ Beard.
Burton-on-Trent	8	3	1	2 12 8	29 0 0	62 12 8	Mr. Power.
Bury	6	3	—	23 12 0	36 7 0	85 14 0	Dr. Beard.

Union.	No. of Vaccination Districts in the Union.	No. of respective Vaccination Contractors recommended for Award.		Range of Awards in each Union.		Total Sum awarded in the Union.	Inspector.
		1st Class Award.	2nd Class Award.	Minimum.	Maximum.		
				£ s. d.	£ s. d.	£ s. d.	
Calne	1	—	—	—	—	—	Dr. Blaxall.
Camberwell	5	5	—	2 16 0	70 12 0	216 2 0	„ Stevens.
Cambridge	1	—	—	—	—	—	„ Airy.
Cannock	4	4	—	1 17 0	48 9 0	70 9 0	Mr. Power.
Canterbury	1	1	—	—	—	27 18 0	Dr. Stevens.
Cardiff	6	5	—	2 4 0	55 12 0	155 17 0	„ Airy.
Cardigan	3	3	—	12 18 0	13 12 0	39 11 0	Do.
Carmarthen	8	3	—	10 10 0	16 8 0	40 10 0	Do.
Carnarvon	5	—	1	—	—	13 3 4	„ Parsons.
Caxton and Arrington	4	4	—	3 12 0	10 6 0	31 2 0	„ Airy.
Cerne	4	1	—	—	—	1 1 0	„ Blaxall.
Chailey	4	2	—	0 16 0	8 12 0	9 8 0	„ Stevens.
Chard	7	1	—	—	—	16 0 0	„ Blaxall.
Cheadle	5	3	—	8 8 0	14 11 1	31 12 0	Mr. Power.
Chelmsford	10	6	—	2 14 0	11 15 0	89 6 0	Dr. Airy.
Chelsea	2	2	—	22 15 0	79 8 0	102 3 0	„ Stevens.
Chester-le-Street	2	2	—	52 17 0	82 16 0	135 13 0	Mr. Power.
Chesterton	7	5	—	7 13 0	12 5 0	49 3 0	Dr. Airy.
Chippenham	7	2	1	3 15 0	15 17 0	23 10 0	„ Blaxall.
Chipping Norton	4	2	—	5 9 0	9 1 0	14 10 0	„ Thorne.
Chorley	5	4	—	7 19 0	34 9 0	81 13 0	„ Beard.
Church Stretton	4	2	—	3 14 0	4 6 0	8 0 0	„ Blaxall.
Cleobury Mortimer	2	1	—	—	—	6 4 0	Do.
Clitheroe	5	—	—	—	—	—	„ Beard.
Clun	4	2	—	6 8 0	6 8 0	12 16 0	„ Blaxall.
Colchester	1	1	—	—	—	40 19 0	„ Airy.
Conway	2	—	—	—	—	—	„ Parsons.
Corwen	3	—	—	—	—	—	Do.
Cranbrook	6	3	2	1 1 4	6 11 0	17 3 4	„ Stevens.
Crickhowell	2	1	1	12 17 0	21 8 0	34 5 0	„ Airy.
Cricklade and Wootton Bassett.	3	1	—	—	—	7 5 0	„ Blaxall.
Cuckfield	6	5	—	2 10 0	9 6 0	36 13 0	„ Stevens.
Darlington	5	2	—	6 13 0	63 9 0	70 2 0	Mr. Power.
Dartford	5	5	—	13 3 0	25 16 0	93 2 0	Dr. Stevens.
Daventry	6	4	—	1 8 0	12 5 0	27 11 0	„ Thorne.
Devises	6	3	—	2 14 0	15 17 0	27 15 0	„ Blaxall.
Dewsbury	11	5	—	7 17 0	21 17 0	70 12 0	„ Beard.
Dolgelly	5	2	—	7 19 0	10 8 0	18 7 0	„ Parsons.
Doncaster	7	3	—	3 1 0	31 6 0	41 17 0	„ Beard.
Dorchester	6	2	—	5 15 0	6 6 0	12 1 0	„ Blaxall.
Dover	4	1	—	—	—	25 14 0	„ Stevens.
Drayton	5	1	1	1 16 8	4 18 0	6 14 8	„ Blaxall.
Dulverton	3	2	—	1 0 0	1 14 0	2 14 0	Do.
Dunmow	6	3	—	7 16 0	11 6 0	28 12 0	„ Airy.
Durham	5	2	—	26 5 0	39 16 0	66 1 0	Mr. Power.
Easington	5	4	—	22 16 0	42 5 0	116 7 0	Do.
Easingwold	4	3	—	4 8 0	6 1 0	16 0 0	Do.
Eastbourne	4	4	—	1 16 0	30 11 0	38 15 0	Dr. Stevens.
East Grinstead	6	2	—	4 15 0	8 1 0	12 16 0	Do.
East Preston	3	2	—	11 5 0	15 12 0	26 17 0	Do.
Eastry	7	6	—	1 17 0	10 3 0	40 1 0	Do.
Ecclesall Bierlow	3	1	—	—	—	70 12 0	„ Beard.
Elham	5	2	—	5 0 0	6 14 0	11 14 0	„ Stevens.
Ellesmere	7	1	1	1 16 0	2 13 0	4 9 0	„ Blaxall.
Ely	7	3	—	0 3 0	9 0 0	17 17 0	„ Airy.
Epping	10	7	—	1 5 0	9 9 0	33 12 0	Do.
Eton	8	4	—	3 12 0	8 18 0	25 7 0	„ Thorne.
Exeter	1	1	—	—	—	39 13 0	„ Stevens.
Faversham	4	4	—	2 9 0	34 9 0	59 10 0	Do.
Festiniog	4	—	—	—	—	—	„ Parsons.
Foleshill	5	1	1	3 16 8	16 2 0	19 18 8	Mr. Power.
Forden	4	1	—	—	—	8 14 0	Dr. Parsons.
Fylde, The	5	4	—	9 19 0	25 2 0	65 6 0	„ Beard.

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Inspection of Public Vaccination.

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		1st Class Award.	2nd Class Award.	Minimum.	Maximum.		
				£ s. d.	£ s. d.	£ s. d.	
Garstang	3	2	—	9 19 0	12 10 0	22 9 0	Dr. Beard.
Gateshead	5	1	—	—	—	24 17 0	Mr. Power.
George, St., East	1	1	—	—	—	79 17 0	Dr. Stevens.
Goole	5	2	—	2 19 0	7 4 0	10 3 0	„ Beard.
Gower	2	1	—	—	—	11 0 0	„ Airy.
Gravesend and Milton	1	1	—	—	—	28 18 0	„ Stevens.
Guisborough	4	1	—	—	—	25 14 0	Mr. Power.
Hackney	4	4	—	13 7 0	66 18 0	152 11 0	Dr. Stevens.
Hailsham	7	4	—	3 1 0	5 1 0	15 17 0	Do.
Halifax	9	5	—	12 16 0	112 0 0	178 13 0	„ Beard.
Halstead	5	4	—	4 10 0	13 9 0	32 13 0	„ Airy.
Hardingstone	3	2	—	7 5 0	9 3 0	16 8 0	„ Thorne.
Hartlepool	3	3	—	3 4 0	55 10 0	98 12 0	Mr. Power.
Haslingden	5	4	—	11 3 0	40 16 0	102 2 0	Dr. Beard.
Hastings	2	1	1	2 1 4	51 12 0	53 13 4	„ Stevens.
Haverfordwest	4	—	—	—	—	—	„ Airy.
Hawarden	3	2	—	17 12 0	20 8 0	38 0 0	„ Parsons.
Hay	3	2	—	4 0 0	10 4 0	14 4 0	„ Airy.
Headington	2	2	—	10 8 0	39 3 0	49 11 0	„ Thorne.
Helmsley Blackmoor	2	2	—	5 4 0	8 7 0	13 11 0	Mr. Power.
Hemsworth	4	2	—	2 1 0	7 7 0	9 8 0	Dr. Beard.
Henley	6	3	—	4 15 0	8 7 0	18 12 0	„ Thorne.
Highworth & Swindon	4	—	—	—	—	—	„ Blaxall.
Holbeck	1	—	—	—	—	—	„ Beard.
Hollinghourn	6	4	—	2 14 0	9 2 0	23 6 0	„ Stevens.
Holyhead	3	—	—	—	—	—	„ Parsons.
Holywell	5	2	1	3 12 0	40 6 0	74 15 0	Do.
Hoo	1	1	—	—	—	5 3 0	„ Stevens.
Horsham	6	6	—	2 18 0	19 17 0	44 15 0	Do.
Houghton-le-Spring	3	3	—	19 13 0	23 18 0	64 16 0	Mr. Power.
Howden	4	4	—	4 11 0	9 18 0	28 17 0	Dr. Beard.
Huddersfield	16	9	—	4 0 0	31 6 0	151 2 0	Do.
Hunslet	3	3	—	2 3 0	53 13 0	63 13 0	Do.
Huntingdon	5	4	—	5 19 0	9 15 0	23 14 0	„ Airy.
Islington	4	4	—	35 5 0	70 15 0	221 6 0	„ Stevens.
Ives, St.	5	3	1	1 12 0	11 0 0	24 18 8	„ Airy.
Keighley	4	1	—	—	—	4 13 0	„ Beard.
Kendal	8	7	—	3 17 0	14 13 0	59 14 0	Mr. Power.
Kettering	4	3	—	11 4 0	33 18 0	65 9 0	Dr. Thorne.
Kirkby Moorside	1	1	—	—	—	8 13 0	Mr. Power.
Knaresborough	3	2	—	7 15 0	12 19 0	20 14 0	Dr. Beard.
Knighton	5	1	—	—	—	6 12 0	„ Airy.
Lambeth	5	4	—	20 4 0	96 4 0	251 3 0	„ Stevens.
Lampeter	2	1	—	—	—	12 13 0	„ Airy.
Lancaster	5	1	—	—	—	9 2 0	„ Beard.
Lanchester	4	3	1	6 14 0	58 10 0	109 3 4	Mr. Power.
Langport	6	—	—	—	—	—	Dr. Blaxall.
Leeds	7	7	—	2 3 0	55 13 0	167 11 0	„ Stevens.
Leek	5	3	—	7 8 0	33 18 0	55 5 0	Mr. Power.
Leigh	3	1	—	—	—	40 12 0	Dr. Beard.
Lewes	1	1	—	—	—	12 8 0	„ Stevens.
Lewisham	5	5	—	7 6 0	21 3 0	71 2 0	Do.
Lexden and Winstree	10	3	—	3 11 0	9 3 0	18 2 0	„ Airy.
Leyburn	6	2	—	1 17 0	3 5 0	5 2 0	Mr. Power.
Lichfield	6	3	1	2 14 0	24 10 0	49 11 0	Do.
Linton	3	—	—	—	—	—	Dr. Airy.
Liverpool	3	2	—	81 18 0	102 8 9	184 6 0	„ Stevens.
Llandilo Fawr	4	2	1	1 2 0	21 14 0	39 8 0	„ Airy.
Llandovery	2	—	—	—	—	—	Do.
Llanelly	3	2	—	25 15 0	79 9 0	105 4 0	Do.
Llanfyllin	5	1	—	—	—	4 0 0	„ Parsons.
Llanwrst	2	—	—	—	—	—	Do.
London, City	2	—	—	—	—	—	„ Stevens.
Ludlow	5	—	1	—	—	3 4 8	„ Blaxall.
Lunesdale	5	3	—	1 4 0	6 7 0	8 19 0	„ Beard.

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		1st Class Award.	2nd Class Award.	Minimum.	Maximum.		
				£ s. d.	£ s. d.	£ s. d.	
Machynlleth	5	—	—	—	—	—	Dr. Parsons.
Madeley	4	1	—	—	—	33 18 0	„ Blaxall.
Maidstone	5	3	—	8 11 0	62 6 0	84 2 0	„ Stevens.
Maldon	7	4	—	6 5 0	12 6 0	35 9 0	„ Airy.
Malling	4	4	—	11 17 0	19 10 0	60 15 0	„ Stevens.
Malmesbury	4	1	—	—	—	6 17 0	„ Blaxall.
Malton	6	3	—	5 8 0	6 0 0	17 5 0	Mr. Power.
Manchester	3	3	—	47 7 0	62 18 0	170 9 0	Dr. Stevens.
Marlborough	2	1	—	—	—	4 7 0	„ Blaxall.
Marylebone	1	1	—	—	—	154 14 0	„ Stevens.
Medway	3	3	—	31 10 0	46 1 0	115 14 0	Do.
Melksham	2	2	—	11 17 0	19 7 0	31 4 0	„ Blaxall.
Mere	2	—	1	—	—	5 7 4	Do.
Meriden	4	4	—	1 15 0	10 18 0	19 15 0	Mr. Power.
Merthyr Tydvil	9	8	—	5 0 0	63 4 0	235 0 0	Dr. Airy.
Middlesbrough	4	3	—	9 17 0	123 4 0	176 10 0	Mr. Power.
Midhurst	5	2	—	4 16 0	12 17 0	17 13 0	Dr. Stevens.
Mile End Old Town	2	1	—	—	—	83 0 0	Do.
Milton	2	1	—	—	—	19 0 0	Do.
Narberth	4	—	—	—	—	—	„ Airy.
Neath	7	1	1	6 12 8	21 6 0	27 18 8	Do.
Neot's, St.	6	5	—	0 13 0	16 11 0	35 2 0	Do.
Newcastle-in-Emlyn	3	—	—	—	—	—	Do.
Newcastle-on-Tyne	3	4	—	26 8 0	51 13 0	140 16 0	„ Stevens.
Newctle.-under-Lyme	3	1	—	—	—	41 0 0	Mr. Power.
Newhaven	5	3	—	0 14 0	10 17 0	14 7 0	Dr. Stevens.
Newmarket	8	4	1	4 4 8	12 18 0	46 2 8	„ Airy.
Newport (Salop)	4	—	—	—	—	—	„ Blaxall.
Newport Pagnell	10	4	—	1 11 0	18 3 0	33 13 0	„ Thorne.
Newtown & Llanidloes	4	—	—	—	—	—	„ Parsons.
Northallerton	4	1	—	—	—	1 8 0	Mr. Power.
Northampton	4	1	—	—	—	14 7 0	Dr. Thorne.
North Aylesford	2	2	—	24 4 0	28 9 0	52 13 0	„ Stevens.
North Bierley	12	4	—	5 2 0	27 10 0	59 17 0	„ Beard.
North Witchford	4	3	—	6 3 0	15 18 0	30 17 0	„ Airy.
Nuneaton	2	—	—	—	—	—	Mr. Power.
Oakham	3	2	—	4 0 0	12 9 0	16 9 0	Dr. Thorne.
Oldham	6	4	—	11 10 0	44 11 0	103 19 0	„ Beard.
Ongar	6	2	—	4 4 0	5 14 0	9 18 0	„ Airy.
Ormskirk	7	6	—	6 6 0	41 12 0	94 9 0	„ Beard.
Orsett	6	2	—	2 18 0	12 7 0	15 5 0	„ Airy.
Oswestry	5	—	1	—	—	5 10 8	„ Blaxall.
Oundle	4	4	—	4 17 0	10 3 0	29 7 0	„ Thorne.
Ouseburn, Great	6	5	—	1 13 0	7 19 0	20 5 0	„ Beard.
Oxford	1	1	—	—	—	33 11 0	„ Thorne.
Paddington	2	2	—	36 1 0	43 0 0	79 1 0	„ Stevens.
Pancras, St.	1	1	—	—	—	293 1 0	Do.
Pateley Bridge	3	1	—	—	—	2 15 0	„ Beard.
Pembroke	6	5	—	7 0 0	23 16 0	65 8 0	„ Airy.
Penistone	3	3	—	8 5 0	17 16 0	38 2 0	„ Beard.
Peterborough	7	3	1	1 19 0	55 19 0	71 11 0	„ Thorne.
Petworth	4	3	—	3 2 0	8 12 0	18 4 0	„ Stevens.
Pewsey	4	3	—	6 18 0	10 11 0	25 18 0	„ Blaxall.
Pickering	3	—	—	—	—	—	Mr. Power.
Pocklington	6	3	—	2 18 0	5 12 0	12 14 0	Dr. Beard.
Pontardawe	2	2	—	34 9 0	35 2 0	69 11 0	„ Airy.
Pontefract	7	7	—	3 2 0	30 3 0	86 9 0	„ Beard.
Pontypridd	8	4	—	17 15 0	55 1 0	113 1 0	„ Airy.
Poole	4	2	1	4 0 0	8 14 0	21 2 0	„ Blaxall.
Potterspury	4	—	—	—	—	—	„ Thorne.
Prescot	8	4	—	32 15 0	71 18 0	216 13 0	„ Beard.
Preston	5	2	—	17 11 0	49 17 0	67 8 0	Do.
Prestwich	6	2	1	13 6 0	87 2 0	159 14 0	Do.
Pwllheli	5	2	—	7 9 0	9 3 0	16 12 0	„ Parsons.
Reeth	2	1	—	—	—	7 13 0	Mr. Power.
Rhayader	2	—	—	—	—	—	Dr. Airy.
Richmond (N. York)	5	4	—	2 0 0	6 9 0	17 12 0	Mr. Power

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				£ s. d.	£ s. d.	£ s. d.	
Ripon	4	2	—	3 4 0	5 1 0	8 5 0	Dr. Beard.
Rochdale	6	5	—	3 0 0	88 17 0	139 12 0	Do.
Rochford	5	1	—	—	—	10 7 0	„ Airy.
Romford	7	2	2	1 6 0	32 1 0	59 11 8	Do.
Romney Marsh	3	3	—	2 0 0	5 19 0	10 18 0	„ Stevens.
Rotherham	8	4	—	3 7 0	59 11 0	114 17 0	„ Beard.
Rugby	8	—	—	—	—	—	Mr. Power.
Ruthin	3	—	—	—	—	—	Dr. Parsons.
Rye	3	2	—	3 3 0	14 8 0	17 11 0	„ Stevens.
Saddleworth	1	—	—	—	—	—	„ Beard.
Saffron Walden	7	4	—	1 19 0	16 16 0	37 12 0	„ Airy.
Saviour, St.	5	5	—	30 6 0	76 2 0	279 13 0	„ Stevens.
Scarborough	5	3	—	3 14 0	33 14 0	45 10 0	Mr. Power.
Sedbergh	2	1	—	—	—	3 5 0	Dr. Beard.
Sedgefield	4	1	—	—	—	11 6 0	Mr. Power.
Seisdon	4	4	—	4 8 0	10 6 0	32 12 0	Do.
Selby	5	4	—	0 18 0	22 12 0	28 4 0	Dr. Beard.
Settle	7	3	—	0 8 0	11 0 0	14 17 0	Do.
Sevenoaks	6	3	—	3 6 0	8 2 0	18 11 0	„ Stevens.
Shaftesbury	3	1	—	—	—	6 8 0	„ Blaxall.
Sheffield	6	5	—	11 14 0	55 19 0	172 3 0	„ Stevens.
Sheppey	2	2	—	5 0 0	28 8 0	33 8 0	Do.
Shepton Mallet	6	1	—	—	—	14 4 0	„ Blaxall.
Sherborne	3	1	1	6 8 0	7 7 4	13 15 4	Do.
Shifnal	4	2	—	3 13 0	6 14 0	10 7 0	Do.
Skipton	8	4	—	1 12 0	10 9 0	22 7 0	„ Beard.
Solihull	4	3	—	7 4 0	9 17 0	25 3 0	Mr. Power.
Southam	4	3	—	4 7 0	9 17 0	21 8 0	Do.
South Shields	3	2	—	51 14 0	78 17 0	130 11 0	Do.
Stafford	4	4	—	5 4 0	25 5 0	58 17 0	Do.
Steyning	5	4	—	6 10 0	23 2 0	48 16 0	Dr. Stevens.
Stockton	3	2	—	5 16 0	52 19 0	58 15 0	Mr. Power.
Stokesley	4	1	—	—	—	9 12 0	Do.
Stoke-upon-Trent	5	3	—	9 4 0	37 0 0	81 4 0	Do.
Stone	4	2	—	9 6 0	16 4 0	25 10 0	Do.
Stratford-on-Avon	7	2	—	3 15 0	15 6 0	19 1 0	Do.
Sturminster	4	2	—	4 18 0	8 13 0	13 11 0	Dr. Blaxall.
Sunderland	5	4	—	27 4 0	81 12 0	224 12 0	Mr. Power.
Swansea	3	3	—	32 10 0	130 11 0	271 3 0	Dr. Airy.
Tadcaster	4	—	—	—	—	—	„ Beard.
Tamworth	3	2	—	9 16 0	20 13 0	30 9 0	Mr. Power.
Taunton	8	5	—	5 16 0	23 3 0	53 19 0	Dr. Blaxall.
Teesdale	7	2	—	3 5 0	15 7 0	18 12 0	Mr. Power.
Tendring	10	1	—	—	—	4 2 0	Dr. Airy.
Tenterden	6	2	—	4 4 0	7 15 0	11 19 0	„ Stevens.
Thakeham	2	2	—	9 19 0	11 12 0	21 11 0	Do.
Thame	6	5	—	0 9 0	10 0 0	28 19 0	„ Thorne.
Thanet, Isle of	4	1	—	—	—	12 9 0	„ Stevens.
Thirsk	6	4	—	2 1 0	4 8 0	11 0 0	Mr. Power.
Thorne	6	2	—	2 9 0	3 7 0	5 16 0	Dr. Beard.
Thrapston	5	4	—	2 9 0	13 18 0	30 15 0	„ Thorne.
Ticehurst	8	5	—	1 1 0	10 12 0	28 4 0	„ Stevens.
Tisbury	3	2	1	5 1 0	5 5 4	15 10 4	„ Blaxall.
Todmorden	2	2	—	14 9 0	17 6 0	31 15 0	„ Beard.
Tonbridge	6	6	—	6 2 0	23 10 0	86 5 0	„ Stevens.
Towcester	4	2	—	6 16 0	12 10 0	19 6 0	„ Thorne.
Tregaron	2	—	—	—	—	—	„ Airy.
Uckfield	6	6	—	1 13 0	12 12 0	54 11 0	„ Stevens.
Ulverstone	7	3	—	3 0 0	31 2 0	42 0 0	Mr. Power.
Uttoxeter	5	1	1	3 8 8	5 8 0	8 16 8	Do.
Wakefield	6	4	—	7 2 0	60 3 0	124 9 0	Dr. Beard.
Walsall	4	3	—	18 0 0	94 12 0	150 18 0	Mr. Power.
Wandsworth and Clapham.	8	4	—	29 4 0	110 12 0	244 14 0	Dr. Stevens.
Wareham and Purbeck.	7	1	—	—	—	5 19 0	„ Blaxall.

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				£ s. d.	£ s. d.	£ s. d.	
Warminster	5	2	—	2 17 0	9 16 0	12 13 0	Dr. Blaxall.
Warrington	3	2	—	33 9 0	101 6 0	134 15 0	„ Beard.
Warwick	3	1	—	—	—	16 12 0	Mr. Power.
Weardale	5	4	—	3 8 0	20 11 0	47 14 0	Do.
Wellingborough	4	3	—	8 16 0	21 5 0	39 14 0	Dr. Thorne.
Wellington (Salop)	3	2	—	20 8 0	39 18 0	60 6 0	„ Blaxall.
Wellington (Somerset)	6	1	—	—	—	9 0 0	Do.
Wem	4	3	—	6 3 0	6 19 0	20 1 0	Do.
West Bromwich	5	3	—	15 13 0	57 1 0	108 6 0	Mr. Power.
Westbury and Whorwelsdown.	4	1	1	1 8 8	19 10 0	20 18 8	Dr. Blaxall.
West Derby	6	1	—	—	—	40 6 0	„ Beard.
West Firle	3	2	1	1 4 0	1 11 0	4 1 0	„ Stevens.
West Ham	9	3	—	23 8 0	87 0 0	167 9 0	„ Airy.
Wetherby	6	2	—	2 11 0	4 6 0	6 19 0	„ Beard
Weymouth	6	3	—	4 7 0	19 12 0	31 5 0	„ Blaxall.
Wharfedale	3	2	—	16 10 0	18 11 0	35 1 0	„ Beard.
Whitby	4	1	1	2 4 8	10 18 0	13 2 8	Mr. Power.
Whitchurch (Salop)	4	3	—	1 14 0	12 9 0	22 0 0	Dr. Blaxall.
Whittlesey	2	2	—	6 19 0	7 16 0	14 15 0	„ Airy.
Wigan	8	4	—	17 4 0	50 19 0	159 3 0	„ Beard.
Williton	7	2	—	6 3 0	6 14 0	12 17 0	„ Blaxall.
Wilton	4	—	—	—	—	—	Do.
Wimborne and Cranbourne.	4	2	—	6 1 0	10 4 0	16 5 0	Do.
Wincanton	6	—	—	—	—	—	Do.
Winslow	3	2	—	6 19 0	7 1 0	14 0 0	„ Thorne.
Wisbeach	14	7	1	1 16 0	13 15 0	51 1 8	„ Airy.
Witham	3	—	—	—	—	—	Do.
Witney	4	3	—	13 16 0	16 2 0	45 8 0	„ Thorne.
Wolstanton and Burslem.	3	3	—	56 4 0	75 0 0	194 7 0	Mr. Power.
Wolverhampton	5	4	—	45 4 0	67 11 0	217 8 0	Do.
Woodstock	5	4	—	4 6 0	7 3 0	22 3 0	Dr. Thorne.
Woolwich	3	1	—	—	—	47 8 0	„ Stevens.
Wortley	3	1	—	—	—	16 19 0	„ Beard.
Wrexham	5	3	—	9 17 0	55 13 0	90 8 0	„ Parsons.
Wycombe	12	8	1	1 8 0	12 2 0	60 15 4	„ Thorne.
Yeovil	7	2	—	2 4 0	7 13 0	9 17 0	„ Blaxall.
Total	1,616	872	42	—	—	16,906 17 4	

No. 3.

App. A. No. 3.

National Vaccine Establishment.

Statistics of the National Vaccine Establishment and Educational Vaccinating Stations.

I.—Staff at end of 1879.

N.B.—The stations named in *italics* are Educational Vaccinating Stations, authorised by the Local Government Board.

—	Name.	Vaccinating Stations.	Days and Hours of Attendance.
Vaccinators supplying lymph for the public service, & salaried from the Parliamentary grant.	1. Dr. R. Cory	*Surrey Chapel*	Tues., Thurs.; 1.
	2. Mr. W. E. G. Pearse	*Tottenham Court Chapel.*	Mon., Wednes.; 1.
Parochial and other Vaccinators not salaried from the Parliamentary grant, but contributing lymph at a fixed rate of payment.	1. Mr. Ellis S. Guest	*Manchester*	Monday; 2.
	2–6. Dr. Edmund Robinson.	*Birmingham*	2. Monday; 3. Tuesday; 4. Wednesday; 11. 5. Wednesday; 2. 6. Thursday; 11.
	7. Mr. Roger Parker	*Liverpool*	Tuesday; 2.30.
	8. Mr. E. L. Webb	*Pimlico*	Thursday; 10.
	9. Mr. J. Hawthorn	*Newcastle-on-Tyne.*	Wednesday; 3.
	10. Mr. W. E. G. Pearse	*Westminster*	Tuesday; 2.
	11–12. Mr. W. A. Sumner	*Marylebone*	Tuesday; 2. Wednesday; 10.
	13. Mr. C. T. Blackman	*Whitechapel*	Wednesday; 11.
	14. Mr. Frederick Holmes	*Leeds*	Tuesday; 2.30.
	15. Dr. Edward Lynes	Coventry	Tuesday; 12.
	16. Dr. Hugh Thomson	*Glasgow*	Monday; 12.
	17. Mr. C. Harriot Roper	*Exeter*	Thursday; 3.
	18. Mr. Robert W. Dunn	*Strand*	Thursday; 11.
	19. Mr. Matthew Brownfield	Bromley, Middlesex.	Tuesday; 3.
	20. Mr. John Broadbent	Ancoats	Wednesday; 2.
	21. Mr. W. Skinner	*Sheffield*	Tuesday; 3.
	22. Dr. A. C. Clarke	Salford	Thursday; 2.
	23. Mr. N. Miskin	Waterloo	Tuesday; 2.
	24. Mr. T. F. Morrish	Toxteth Park	Tuesday; 11.
	25. Dr. J. O. Affleck	*Edinburgh*	Wednes., Sat.; 1.
	26. Dr. R. Cory	*St. Thomas's Hosp.*	Wednesday; 11.30.
	27. Dr. D. C. McVail	*Glasgow West*	Monday; 1.
Teachers of Vaccination not supplying lymph	Dr. W. Husband	*Edinburgh*	Wednes., Sat.; 12
	Dr. R. D. Tannahill	*Glasgow*	Mond., Thurs.; 12
	Mr. H. Lawrence	*Bristol*	Wednesday; 10.
	Mr. J. Gittins	*Horsleydown*	Monday; 2.

II.—Sources and Amount of Lymph Supply in 1879.

N.B.—The Stations named in *italics* are Educational Vaccinating Stations authorised by the Local Government Board.

	Vaccinating Stations.	Number of Vaccinations performed at the Stations respectively.		Number of Charges of Lymph supplied from the Stations respectively.	
		Primary.	Re-vaccinations.	Charged ivory Points.	Charged Tubes each estimated as equal to 10 ivory Points.
Vaccinators salaried from the Parliamentary grant.	1. *Surrey Chapel*	577	47	11,039	—
	2. *Tottenham Court Chapel.*	895	30	164*	809
	Total -	1,472	77	11,203	809
Parochial and other Vaccinators not salaried from the Parliamentary grant, but contributing lymph at a fixed rate of payment.	1. *Manchester* -	1,367	—	—	403
	2–6. *Birmingham* -	5,728	54	—	3,687
	7. *Bristol* - -	347†	—	—	—
	8. *Liverpool* -	1,650	7	—	949
	9. *Pimlico* - -	435	18	—	731
	10. *Newcastle on-Tyne.*	736	—	—	1,036
	11. *Westminster* -	827	7	—	1,045
	12–13. *Marylebone* -	1,821	71	—	841
	14. *Whitechapel* -	1,695	27	—	414
	15. *Leeds* - -	1,162	—	—	243
	16. Coventry -	1,083	4	—	1,727
	17. *Glasgow* - -	1,386	—	—	1,810
	18. *Exeter* - -	635	1	—	—
	19. *Strand* - -	493	16	—	—
	20. Bromley - -	1,409	8	—	548
	21. Ancoats - -	1,193	—	—	4,772
	22. *Sheffield* - -	1,112	—	—	898
	23. Salford - -	863	—	—	930
	24. Waterloo -	655	21	—	1,506
	25. Toxteth Park -	1,723	28	—	2,036
	26. *Edinburgh* -	1,130	7	—	160
	27. *St. Thomas's Hospital.*	602	7	—	610
	28. *Glasgow West*†	689	1	—	275
	Total -	28,741	277	—	24,621
	General Total -	30,213	354	11,203	25,430

* This number includes 41 charged glasses, each estimated as equal to four charged ivory points.

† To the end of July only.

APP. A. No. 3.

National Vaccine Establishment.

III.—DISTRIBUTION OF LYMPH, 1879.

Number of applications:

From Medical Practitioners in England and Wales	8,390
„ „ „ Scotland	213
„ the Army	446
„ the Navy and from the Emigration Department	78
„ India and the Colonies	191
„ Diplomatic and other Foreign Services	92
Total	9,410

Supplies sent out:—

Charged ivory points	13,372
„ squares of glasses	41
„ capillary tubes	25,006

No. 4.

On Results of Vaccination with Animal Lymph obtained from Dr. Warlomont, of Brussels; by Dr. Robert Cory.

On the 23rd of February 1880, I received five points and five tubes of Dr. Warlomont's lymph, which he stated were taken on the 20th of February from a heifer vaccinated on the 14th. The tubes were all perfectly sealed with paraffin. The lymph from one of them was examined on the 9th of March. It was then easily blown out, contained no coagulum, and readily dried. Under the microscope no blood disks were seen, but there were many moving bacteria; the presence, however, of the latter is very common in lymph preserved in tubes. The other four tubes were used for vaccination.

The lymph on the points was blood-stained, and to one point a hair was adhering, which was removed and examined. It was found to measure between $\frac{5}{100}$ and $\frac{6}{100}$ of a millimeter (*see* fig. 3), and was very similar to an ox hair, with which it was compared.

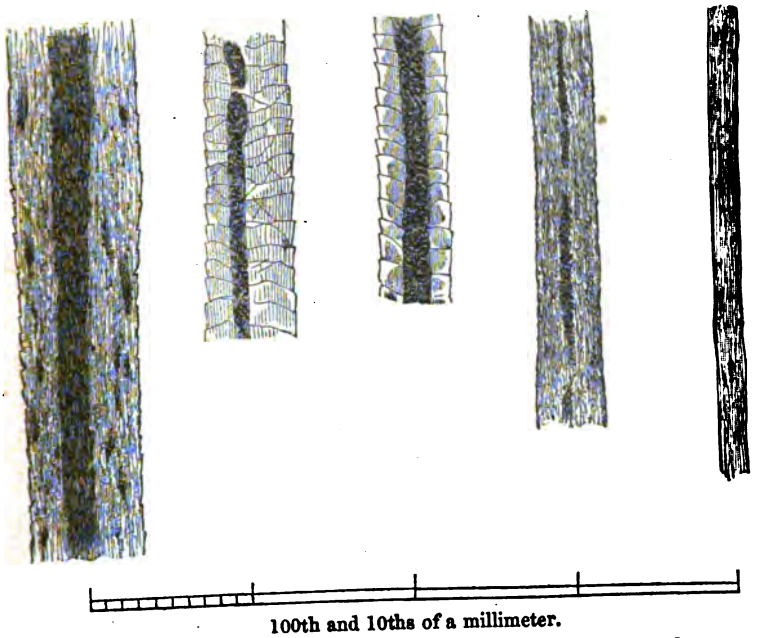
Fig. 1. Hair of young ox.

Fig. 2. Under hair from belly of calf, 3 days old.

Fig. 3. Hair found in Dr. Warlomont's lymph.

Fig. 4. Hair from upper part of adult's arm (human).

Fig. 5. Lanugo from arm of child, 3 months old.



100ths and 10ths of a millimeter.

All the hairs above represented are drawn to the same scale.

Results of Vaccination with the Lymph.

On the 24th February three children, viz., John E. Moore, Nelly Purdye, and William Hawks were vaccinated at Surrey Chapel, each being done with one tube of the lymph. Five insertions were made in every case.

On the 25th February three children, viz., Emily A. Briscoe, Annie Dukes, and William Brine were vaccinated at St. Thomas's Hospital, with two points, two points, and one point respectively of the lymph. Five insertions were made in every case.

In using these points it was noticed that the lymph was only removed from them after considerable rubbing, and that it was only slowly soluble.

On the 2nd of March the children vaccinated at Surrey Chapel with the tubes were inspected. Out of the 15 insertions 11 were successful.

On the 3rd of March the children vaccinated at St. Thomas's Hospital with the points were inspected. All the 15 insertions were successful.

Another child was vaccinated on the 4th of March with a tube of the lymph which had been kept since the 23rd of February. All five insertions were *un*successful.

This is so far corroborative of what Dr. Warlomont himself says of his tube lymph, viz., that it will not keep active for long, but should be used within 24 hours of its reception.

The particulars of the above cases follow :—

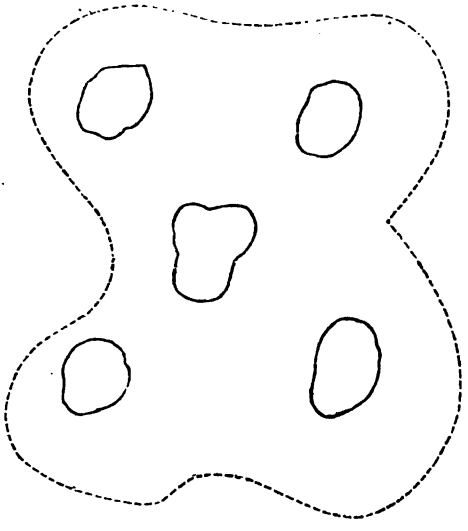
John E. Moore.—Born on September 25, 1879, was vaccinated at Surrey Chapel with one tube of Dr. Warlomont's lymph on the 24th of February (Tuesday).

Inspected on March 2nd. All five places had taken. The vesicles were of fair size. No areola was present.

The child was visited on the 4th of March. The vesicles were then larger than when seen on the 2nd, and the areola was present.

The size of the vesicles and size of areola are indicated in the figure.

Vesicles with their Areolas on the 10th Day.



The child was again seen on March 12th. On that day one scab had then fallen.

On the 18th there were still two scabs adhering. It was ascertained that one scab fell on the 13th.

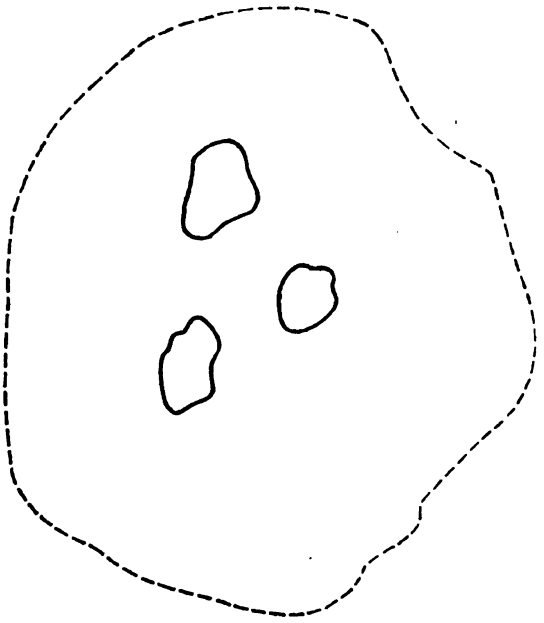
The child was last seen on the 23rd of March, when all the scabs had fallen. The last scab fell on the 20th. Dry secondary scabs were adhering to the places.

Nelly Purdye.—Born on the 22nd November 1879, was vaccinated at Surrey Chapel with one tube of Dr. Warlomont's lymph on the 24th February (Tuesday).

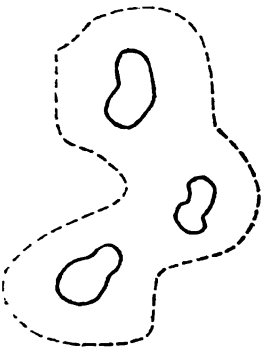
Inspected on 2nd March. Three places had taken. The areola was present. The size of the vesicles and extent of areola are indicated in the left hand figure.

The child was seen again on the 4th March, when the size of the vesicles and extent of areola were as indicated in the right hand figure.

Vesicles with their Areolas on the 10th Day.



Vesicles with their Areolas on the 8th Day.



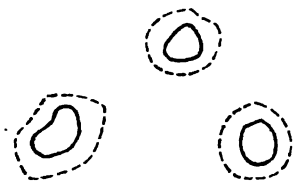
I was unable to see this child after the last-mentioned date on account the mother having moved.

William J. Hawkes.—Born on November 4th, 1879, was vaccinated at Surrey Chapel with one tube of Dr. Warlomont's lymph on the 24th February 1880 (Tuesday).

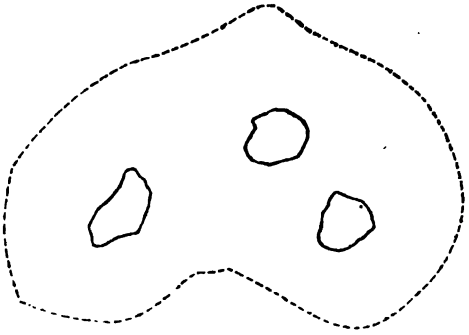
Inspected on March 2nd. Three places had taken. The areola was just commencing as seen in the left hand figure.

The right hand figure represents the size of the vesicles and the extent of the areola as seen on the 4th of March.

Vesicles with their Areolas on the 8th Day.



Vesicles with their Areolas on the 10th Day.



On the 18th of March the child was again seen, and it was then ascertained that the first scab fell on the 11th, and the last on the 13th of March. Dry secondary scabs were adhering to the places.

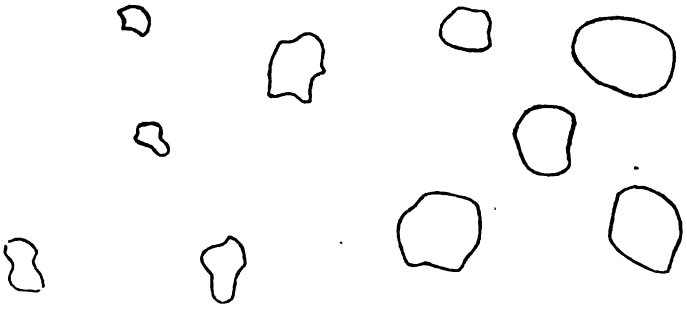
Emily Ann Briscoe.—Born on the 4th of December 1879, was vaccinated at St. Thomas's Hospital with two points of Dr. Warlomont's lymph on the 25th February 1880.

Inspected on the 3rd of March. Five places had taken, but they were small. No areola was present. The left hand figure shows the size of the vesicles.

The child was seen again on the 12th March. The scabs were dry, and firmly adherent. Their size is represented in the right hand figure.

SIZE OF VESICLES ON THE 8TH DAY.

SIZE OF ADHERENT SCABS ON THE 17TH DAY.

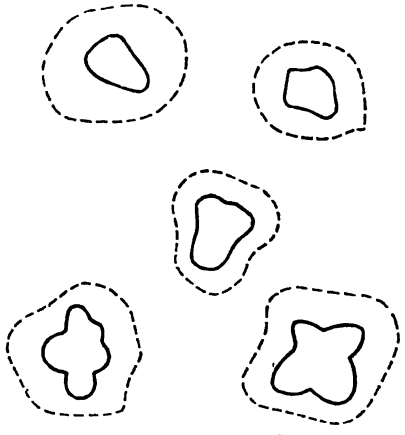


The mother stated the arm was most inflamed on the 5th of March.

Annie Dukes.—Born on the 3rd November 1879, was vaccinated at St. Thomas's Hospital in five places with two points of Dr. Warlomont's lymph on the 25th February 1880.

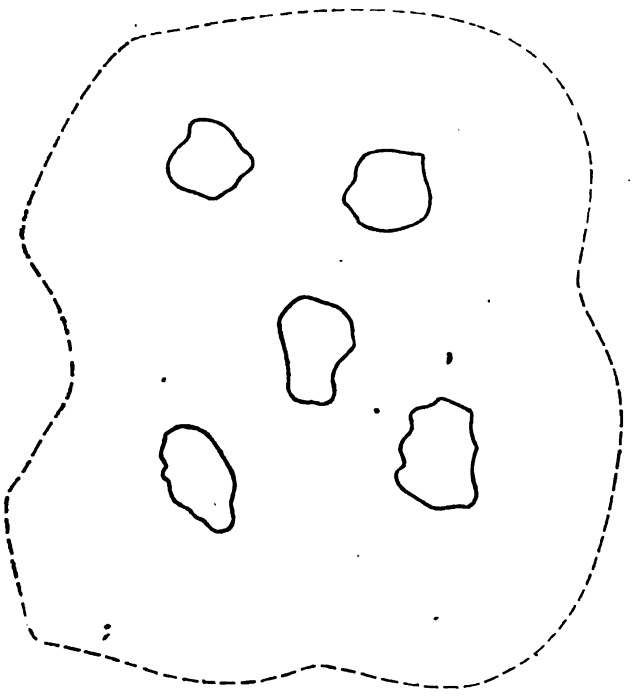
Inspected on the 3rd of March. All five places had taken. The vesicles were of moderate size, and the areola was 2 or 3 mm. in extent. Fig. (*a.*) represents the size of the vesicles and extent of the areola on the 3rd of March.

FIG. (*a.*)—VESICLES WITH THEIR AREOLAS ON THE 8TH DAY.



The child was seen again on the 5th of March 1880. The vesicles had increased in size, and the areola was present to the extent represented in Fig. (*b.*).

Fig. (*b.*)—Vesicles with their Areolas on the 10th Day.



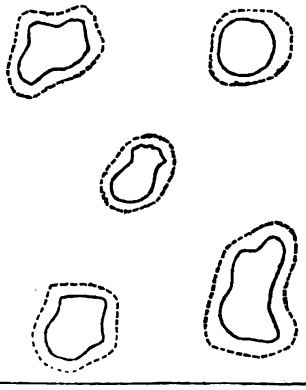
On the 12th of March all the scabs were firmly adherent. The mother states the arm was most red on the 10th, but this cannot have been the case, for there was no redness on the arm on the 12th and the scabs were dry and adherent.

On March 23rd the child was again seen, and it was ascertained that the four last scabs fell on the 16th of March.

William Brine.—Born on the 4th December 1879, was vaccinated at St. Thomas's Hospital from one point of Dr. Warlomont's lymph on the 25th.

Inspected on the 3rd March. Five places had taken.* The areola was just commencing.

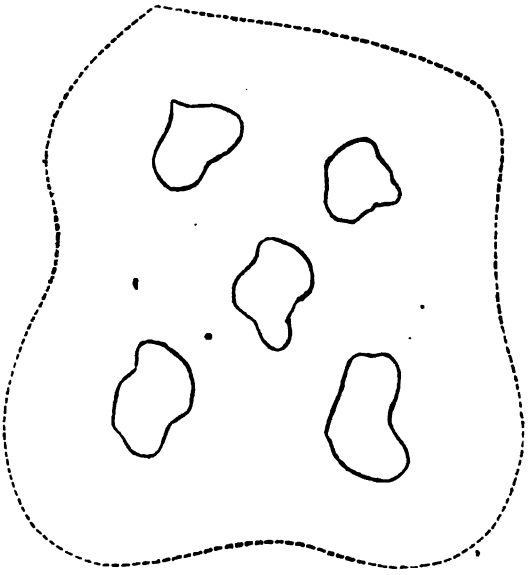
Fig (*c.*)—Vesicles with their Areolas on the 8th Day.



* The mother stated on the 25th of March that she had tried to wash the two lower places off when she got home after the vaccination on the 25th of February.

Fig. (*c.*) represents the vesicles as seen on the 3rd of March, and fig. (*d.*) represents them as seen on the 5th of March.

Fig. (*d.*)—Vesicles with their Areolas on the 10th Day.



On the 12th of March the scabs were all dry but adherent.

On the 23rd of March it was ascertained that one scab fell on the 16th and another on the 22nd.

On the 25th of March the last scab fell. The four places from which the scabs first fell were at this date still discharging.

Results of Vaccination with the Lymph at its First Remove from Dr. Warlomont's stock.

Two children were vaccinated from E. Moore on the 2nd of March. The resulting vesicles were of good size, and the areola extended on the eighth day in one case 8 mm., and in the other 5 mm. All the 10 insertions were successful.

One child was vaccinated from William Hawks on the 2nd of March. The resulting vesicles were of good size, and the areola on the eighth day was 4 mm. in extent. The five insertions were successful.

Three children were vaccinated from Annie Dukes. The resulting vesicles were of good size. All their arms were more or less inflamed. In one case the areola on the eighth day was 9 mm. in extent, in another 17 mm., and in the third 25 mm.

All 15 insertions were successful.

It will be noticed that of the six cases done with the lymph of the first remove from Dr. Warlomont's lymph three were decidedly inflamed on the eighth day. An early areola on the eighth day is what I have before observed in two cases done with lymph at its first remove from a reputed animal source.

Results from the Later Removes of the Lymph.

On the Tuesdays at Surrey Chapel the lymph stock derived from the tubes of Dr. Warlomont was alone used. This will be referred to subsequently as No. 1 stock.

On the Wednesdays at St. Thomas's the lymph stock derived from the points of Dr. Warlomont was used concurrently with the old lymph stocks. This will subsequently be referred to as No. 2 stock.

At about the fourth remove the vesicles produced by No. 1 stock were for the most part small and contained thin watery lymphs. There was likewise a tendency to irregularity in the course of the vaccination. On some arms the areola on the eighth day was excessive, in others it was totally absent. Certainly the number of satisfactory results after the fourth remove was not equal to that obtained from the old stock. This was remarked not only by myself but also by the clerk, Mr. Adams, who has recorded the results of the vaccinations at the station for upwards of seven years.

Not only was there this visible degeneration of the vesicles, but the numerical success was less. Thus the success with the first three removes was 100 per cent., for out of 105 insertions 105 took, whereas in the latter removes, including the fourth, the success was only 94·98 per cent., for out of 95 insertions only 90 took.

Table III. shows the cases through which the lymph descended, by the aid of which the number of vaccinations at each remove can also be readily counted.

Table IV. shows the number of successful insertions in each case.

No. 2 stock was used at St. Thomas's Hospital concurrently, as before stated, with the old stock, which had been long transmitted from arm to arm up to the date of trial. After the first remove I purposely kept myself ignorant at the time of vaccination from which of these two stocks the lymph was derived, but kept the usual record of the source of lymph in each case. I followed my usual plan of selecting the best arms for vaccinating from: thus the fact acquires significance that the animal stock became extinct at the sixth remove.

Table V. shows the descent of No. 2 stock and the old stock together.

Table VI. gives the numerical success of 2 stock.

Table VII. gives the numerical success of the old stock during the period No. 2 stock was being used with it at St. Thomas's Hospital.

If the total number of successful insertions done with the two lymph stocks during the first three removes be compared with that of the later removes, we get the following results:—

	Success during first three removes.		Success during later removes, commencing with the fourth.	
	Insertions.	Success.	Insertions.	Success.
No. 1 stock -	105	105	95	90
No. 2 stock -	60	58	15	8
	165	163	110	98

This gives 98·79 as the insertion success with the lymph derived from the first three removes, and 89·1 per cent. as that with the later removes.

The above facts will perhaps bear the inference that lymph of animal origin is apt to be irregular in its early removes from its source, and that during this period the results of the vaccinations with it are less satisfactory.

This becomes an important question when the propagation of animal lymph is proposed as a means of renewing the lymph stocks of the country.

TABLE I., showing the RESULTS of

Number in Vaccination Register.	Name.	Date of Birth.	Source of Lymph.	Amount of Lymph used.	Number of Insertions.	Date of Lymph taking.	Date of Vaccination.	No. of Successful Insertions.
986, S. C.	John E. Moore	Sept. 25	Dr. Warlomont	1 tube	5	Feb. 20	Feb. 24	5
S. C.	Nellie Purdye	Nov. 22	„	1 „	5	„ 20	„ 24	3
988, S. C.	William J. Hawks	„ 4	„	1 „	5	„ 20	„ 24	3
78, St. T. H.	Emily A. Briscoe	Dec. 4	„	2 points	5	„ 20	„ 25	5
80, St. T. H.	Annie Dukes	Nov. 3	„	2 „	5	„ 20	„ 25	5
84, St. T. H.	William Brine*	Dec. 4	„	1 point	5	„ 20	„ 25	5
S. C.	Florence E. Norris†	Oct. 24	„	1 tube		„ 20	Mar. 4	0

* Child's arm not healed on 28th day after vaccination.

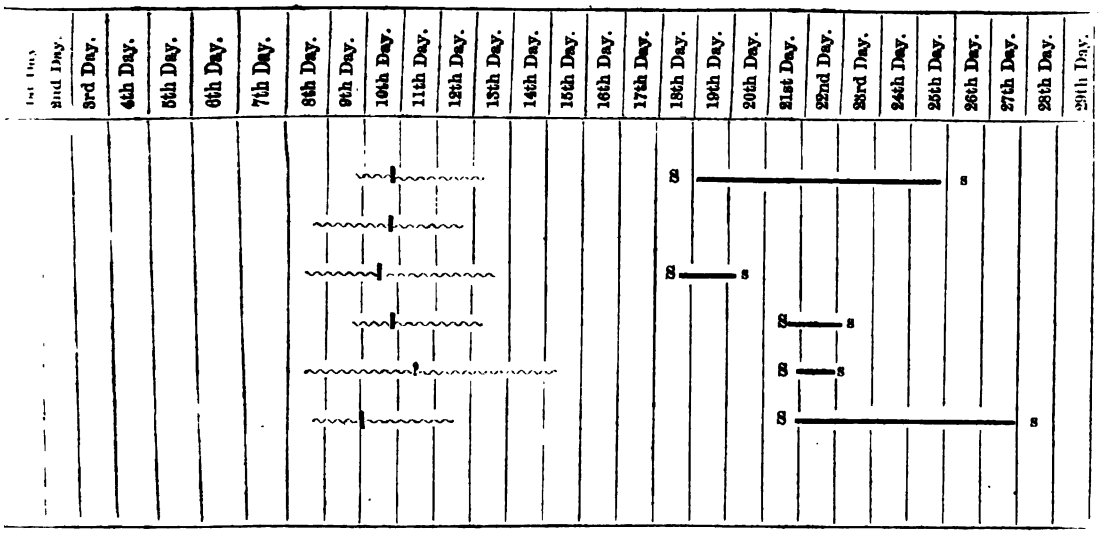
† The mother stated that the child's arm was very red the first two days after the attempted vaccination, and that this redness disappeared on the 3rd day. This child was successfully vaccinated the following week, all the 5 insertions taking.

TABLE II., showing the RESULTS of

Number in Vaccination Register.	Name.	Date of Birth.	Source of Lymph.	Amount of Lymph used.	Number of Insertions.	Date of Lymph taking.	Date of Vaccination.	No. of Successful Insertions.
5, S. C.	Bessie Smithers	Dec. 1879.	988, S. C.	Arm to arm.	5	Mar. 2	Mar. 2	5
5, S. C.	Mary G. Kingstone	Dec. 1879.	986, S. C.	„	5	„ 2	„ 2	5
2, S. C.	Florence Fellows	Oct. 1879.	986, S. C.	„	5	„ 2	„ 2	5
97, St. T. H.	William Hardyman	Sept. 18, 1879.	80, St. T. H.	„	5	„ 3	„ 3	5
98, St. T. H.	Alfred Herbert	Dec. 18, 1879.	80, St. T. H.	„	5	„ 3	„ 3	5
99, St. T. H.	Henry Bender*	Oct. 1879.	80, St. T. H.	„	5	„ 3	„ 3	5
100, St. T. H.	W. Fell (2nd vacc.)	1857	80, St. T. H.	1 tube	4	„ 3	„ 6	4

* This child was brought back with axillary abscess on the 31st March.

VACCINATION done with DR. WARLOMONT'S LYMPH.



The waved line in the above table indicates the day on which the areola commenced, the day of its height, and the time of its decline. Thus, the areola on the arm of John E. Moore commenced on the 9th day after vaccination, was at its height on the 10th day, and had declined by the 13th. S is placed under the day after vaccination on which the first scab fell, and s under the day on which the last one fell.

VACCINATION done with LYMPH of the first remove from above.

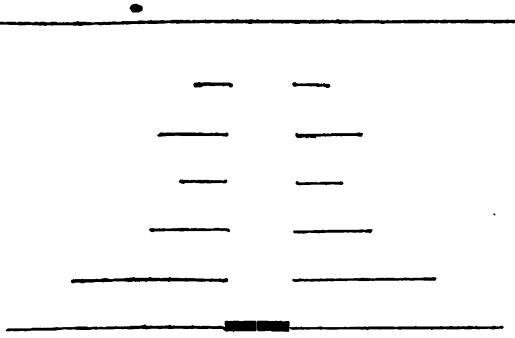
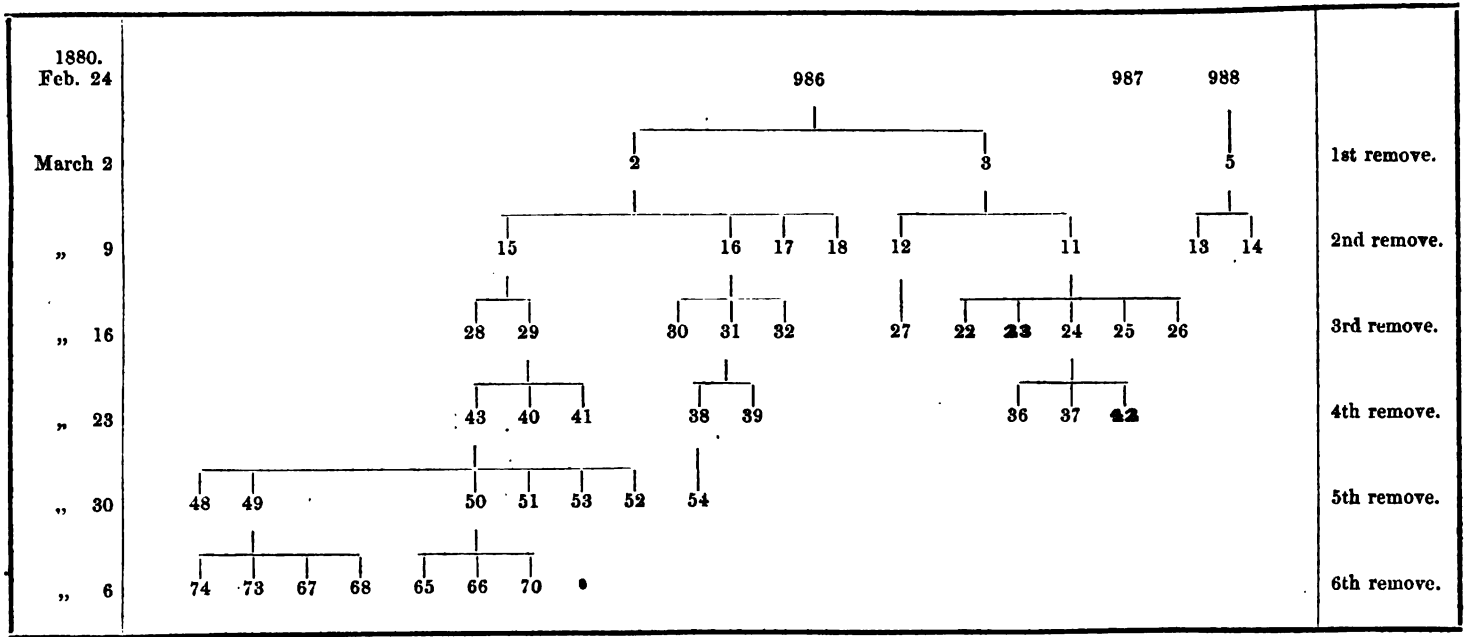


Table III.

Descent of Dr. Warlomont's *Tube* Lymph at Surrey Chapel on *Tuesdays*.



The numbers in the table are those given to the cases in the vaccination register.
The numbers in antique figures were revaccinations.

Table IV.

Table showing the Numerical Success of the Insertions with Lymph in its Descent from Dr. Warlomont's Tube Lymph, used at Surrey Chapel on Tuesdays. No. 1 Stock.

Date 1880.	No. of case.	No. of insertions.	Successful.	—	Date 1880.	No. of case.	No. of insertions.	Successful.	—
Feb. 24	986	.5...	.5...	lymph in tubes.	Mar. 23	43	5	5	—
						50	5	3	—
	987	.5...	.3...	Ditto.		41	5	5	—
	988	.5...	.3...	Ditto.		38	5	5	—
Mar. 2	2	5	5	—		39	5	5	—
	3	5	5	—		36	.5...	.0...	not vacc. by self.
	5	5	5	—					
	15	5	5	—		37	.5...	.0...	Ditto.
	16	5	5	—		42	.5...	.3...	revacc.
	17	5	5	—		48	5	5	—
„ 9	18	5	5	—		49	5	5	—
	12	5	5	—		50	5	5	—
	11	5	5	—	„ 30	51	5	4	—
	13	5	5	—		52	5	5	—
	14	5	5	—		53	5	4	—
	28	5	5	—		54	5	5	—
	29	5	5	—		74	5	5	—
	30	5	5	—		73	5	5	—
	31	5	5	—		67	5	5	—
	32	5	5	—	April 6	68	5	5	—
„ 16	27	5	5	—		65	5	5	—
	22	5	5	—		66	5	4	—
	23	.5...	.?...	revacc.		70	5	5	—
	24	5	5	—			200	195	
	25	5	5	—					
	26	5	5	—					

Equivalent to an insertion success of 97·5 per cent.

N.B.—The first cases done with the preserved lymph, revaccinations and cases not done by myself are represented by figures having dots placed before and after them, and are not included in the total numbers.

Table V.

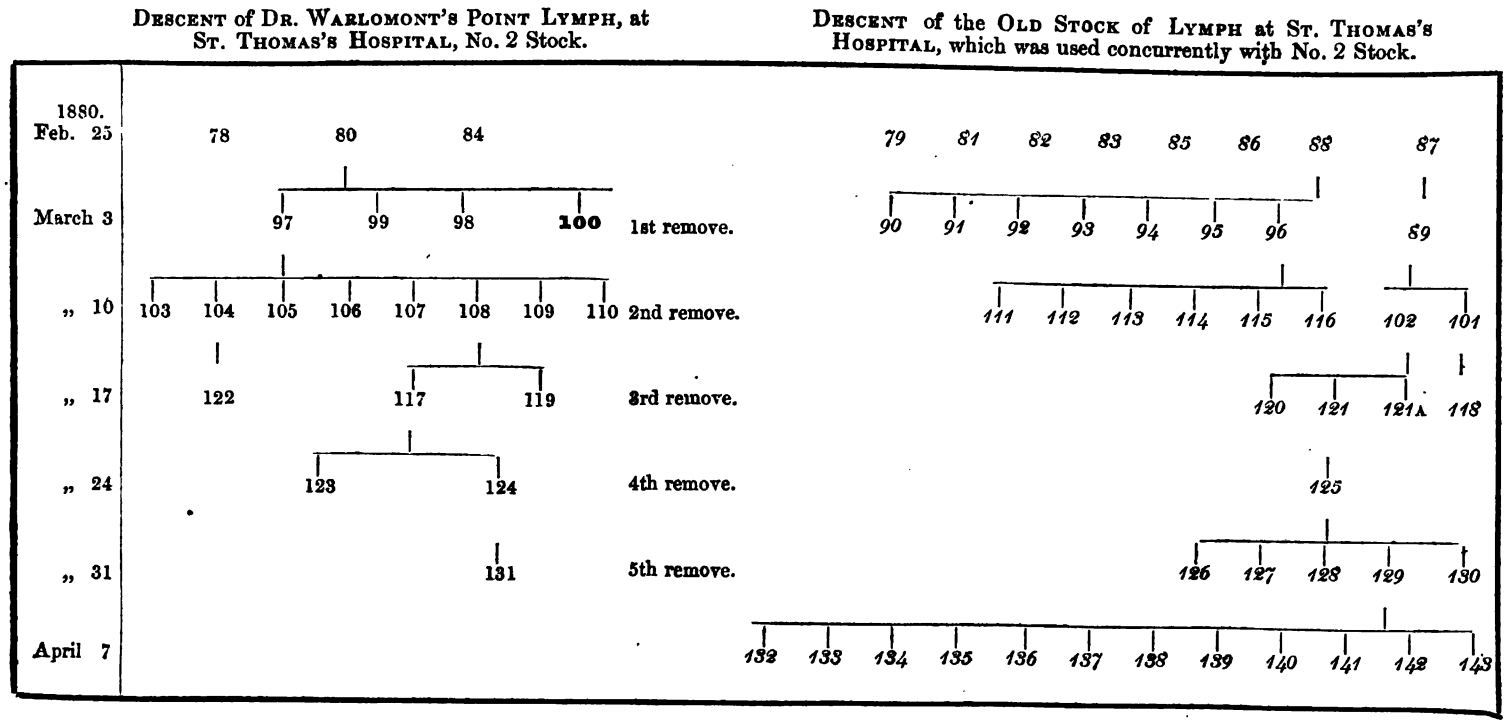

Descent of Dr. Warlomont's Point Lymph, at St. Thomas's Hospital, No. 2 Stock.
Descent of the Old Stock of Lymph at St. Thomas's Hospital, which was used concurrently with No. 2 Stock.
1880.
Feb. 25
March 3
„ 10
„ 17
„ 24
„ 31
April 7
78 80 84
97 99 98 100
1st remove.
103 104 105 106 107 108 109 110
2nd remove.
122 117 119
3rd remove.
123 124
4th remove.
131
5th remove.
79 81 82 83 85 86 88 87
90 91 92 93 94 95 96 89
111 112 113 114 115 116 102 101
120 121 121A 118
125
126 127 128 129 130
132 133 134 135 136 137 138 139 140 141 142 143

Table VI.

Table showing the Numerical Success of the Insertions with Lymph in its Descent from Dr. Warlomont's Point Lymph, used at St. Thomas's Hospital. No. 2 Stock.

Date 1880.	No. of case.	No. of insertions.	Successful.	Remarks.	Date 1880.	No. of case.	No. of insertions.	Successful.	Remarks.
Feb. 25	78	.5...	.5...	points.	Mar. 10	108	.5...	.5...	experimt.
	80	.5...	.5...	points.		109	5	4	—
	84	.5...	.5...	points.		110	5	5	—
Mar. 3	47	5	5	—		117	5	5	—
	48	5	5	—	„ 17	119	.5...	.4...	not done by self.
	49	5	5	—					
	100	.4...	.4...	revacc.		122	5	5	—
„ 10	103	5	5	—	„ 24	123	5	4	—
	104	5	4	—		124	5	3	—
	105	5	5	—	„ 31	131	4	5	—
	106	5	5	—					
	107	5	5	—			75	70	

Equivalent to an insertion success of 93·4 p.c.

Table VII.

Table showing the Numerical Success of Insertions with the Old Stock of Lymph used concurrently at St. Thomas's Hospital with No. 2 Stock.

Date 1880.	No. of case.	No. of insertions.	Successful.	Remarks.	Date 1880.	No. of case.	No. of insertions.	Successful.	Remarks.
Feb. 25	79	5	5	—	Mar. 17	121	5	5	—
	81	5	5	—		121a	5	5	—
	82	5	5	—		120	5	5	—
	83	5	5	—		118	5	5	—
	85	5	4	—	„ 24	125	5	5	—
	86	5	5	—		126	5	5	—
	88	5	5	—		127	5	5	—
	87	5	5	—	„ 31	128	5	4	—
Mar. 3	90	.5...	.5...	not done by self.		129	5	5	—
						130	5	5	—
	91	.5...	.5...	„		132	5	5	—
	92	5	5	—		133	5	5	—
	93	.5...	.5...	not done by self.		134	5	5	—
						135	5	5	—
	94	5	4	—		136	5	5	—
	95	5	5	—	April 7	137	5	5	—
	96	5	5	—		138	5	5	—
	89	5	5	—		139	5	5	—
	111	5	5	—		140	5	4	—
	112	5	5	—		141	5	5	—
	113	5	5	—		142	5	5	—
	114	5	5	—		143	5	5	—
„ 10	115	5	5	—					
	116	5	5	—			215	211	
	102	5	5	—					
	101	5	5	—					

Equivalent to an insertion success of 98·1 p.c.

N.B.—The first cases done with the preserved lymph, revaccinations and cases not done by myself, are represented by figures having dots placed before and after them, and are not included in the total numbers.

No. 5.

ABSTRACT of MEDICAL INSPECTIONS made in 1879 with regard, generally, to the INCIDENCE OF DISEASE on particular places, and to consequent Questions concerning the LOCAL SANITARY ADMINISTRATION.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
1. Bedlingtonshire (Northumberland.) Population, 15,000. *Dr. Parsons.*	Regis.-General's Returns and Reports of Medical Officer of Health.	Bedlingtonshire local board.	Epidemic of scarlatina in a colliery district. Disease mostly spread by personal intercourse, inhabitants being very careless about the propagation of infection. Colliery houses substantially built, but many damp near base of walls. Older houses in an inferior sanitary condition. Courtyards unpaved and in a filthy state. Sewers and house-drains defective in certain respects. Water supply from River Blyth distributed to all parts of district. Large and offensive midden privies in general use. Removal of night-soil and ashes undertaken by colliery owners, but not satisfactorily performed. Slaughter-houses and common lodging-houses ill-regulated. No hospital accommodation for cases of infectious disease, and no disinfecting apparatus.
2. Biddenden - - (Kent.) Population, 380. *Dr. Thorne Thorne.*	Report of Medical Officer of Health.	Tenterden rural sanitary authority.	Frequent recurrence of fatal diphtheria. Water supply scanty, and often polluted. Defective means of drainage, leading to grave nuisance. Privies so constructed and managed as to cause nuisance and injury to health.
3. Bleadon - - - (Somerset.) Population, 411. *Mr. Power.*	Regis.-General's Returns	Axbridge rural sanitary authority.	Extensive and intense prevalence of diphtheria in a small village during a limited time. Defective water supply, drainage, and excrement disposal. No apparent relation between the unsanitary circumstances and the diphtheria. Schools had no appreciable influence in spreading contagion.
4. Chichester - - (Sussex). Population, 7,825. *Dr. Airy.*	Local complaint -	Chichester town council.	Outbreak of enteric fever due to infected milk (but no contamination traced unless it were in the use of water taken from polluted stream for washing the cows' udders). High general death-rate, and sustained prevalence of enteric fever. Drainage mostly into cesspools, often in dangerously close relation with wells, from which a large proportion of the drinking water is obtained. Partly-covered branch of stream receiving sewage foul, stagnant, and insufficiently ventilated. Delay in removal of house refuse, and no system of excrement removal. Medical Officer of Health is also Inspector of Nuisances. Neglect of the sanitary authority to carry out recommendations made by Dr. Seaton in 1865.
5. "Cornwall" Reformatory school-ship. *Mr. Power.*	Request of Home Office for investigation of "fever" outbreak.	- - -	All usual causes of enteric fever excluded. Question discussed of relation of food to the disease. Particulars of examination of exhumed body of the only fatal case. Disproof of enteric fever, and suggestion of trichiniasis having been the disease concerned. (See page 47.)
6. Cranfield and other villages. (Bedfordshire.) *Dr. Cory.*	Regis.-General's Returns	Ampthill rural sanitary authority.	Two separate outbreaks of diphtheria at different villages, one outbreak following the other. Influence of school attendance on spread of disease not established. Water supply imperfect and polluted. Faulty method of excrement disposal.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
7. Crompton (Lancashire.) Population, 8,700. *Dr. Horace Swete.*	Regis.-General's Returns	Crompton local board	Epidemic of scarlatina, and sustained prevalence of enteric fever. General death-rate high, and that from zymotic diseases especially so. Privy accommodation defective, and excremental refuse the source of grave nuisance. Water supplied by waterworks good, but many houses depend on surface wells liable to pollution. Sewer ventilation defective. Law as to nuisances and byelaws not properly enforced. No hospital accommodation or means of disinfection of clothing, &c.
8. Dartford Rural Sanitary District. (Kent.) Population, 25,757. *Dr. Thorne Thorne.*	Communication from Registrar-General.	Dartford rural sanitary authority.	Fatal outbreak of diphtheria in Swanscombe parish, where the conditions of water supply, drainage, and excrement disposal were generally most defective. Sanitary circumstances of the entire rural district also found to be in many respects similar to those at Swanscombe, and sanitary administration very imperfect in many essential respects.
9. Easington Rural Sanitary District. (Durham.) Population, 30,900. *Mr. Power.*	Regis.-General's Returns	Easington rural sanitary authority.	Severe and sustained prevalence of scarlatina in colliery villages, its incidence falling almost entirely on young children. Crowding together of old house property. Dwellings of this sort small, damp, ill-lighted, and ill-ventilated. Arrangements for water supply imperfect or incomplete. Many older colliery villages destitute of privies. Means for disposal of refuse defective. Slop drainage imperfect. Sanitary administration mostly left to colliery owners. Habitual disregard by colliers of precautions against infection. No proper means for isolation of infectious diseases, and absence of public provision for disinfection.
10. Fallowfield, near Manchester. (Lancashire.) *Dr. Airy.*	Local information	Rusholme local board	Sudden outbreak of scarlet fever, attended with vomiting and diarrhœa, among customers of one dairy. Detailed evidence that the fever was spread by the milk supplied by this dairy; that while probably on one or two previous days the milk had been contaminated, on one special occasion it was especially contaminated, and that the period of incubation was about 48 hours. Evidence as to the manner in which the milk became infected inconclusive. (*See* p. 93.)
11. Haverfordwest Rural Sanitary District. (Pembrokeshire.) Population, 24,460. *Dr. Swete.*	Regis.-General's Returns	Haverfordwest rural sanitary authority.	Continuous prevalence of enteric fever, and serious prevalence of scarlatina and diphtheria. Water supply habitually open to surface drainage. Many of the streams polluted by excreta, and wells greatly exposed to pollution. No arrangement for disposal of excrement, few houses having any sort of privy accommodation. Little or no drainage. Grave nuisance from deposits of excrement, mixen heaps, foul pools, and keeping of animals. House accommodation bad and dilapidated. Many houses unfit for habitation. No hospital and no systematic plan of disinfection.
12. Holyhead and Llanddaussaint Registration Sub-districts. (Anglesea.) *Dr. W. Ogle.*	Regis.-General's Returns	Holyhead local board	*Holyhead Urban Sanitary District.* Outbreak of small-pox in an overcrowded lodging-house. No steps taken by local board to secure isolation, although an infectious hospital was available for the purpose. Serious outbreak of diphtheria, the incidence of which fell almost entirely upon children attending a small overpacked dame's school. Sewer of town ill-ventilated and ill-flushed. Privy accommodation of poorer classes very bad. No system of public scavenging. Grave nuisances from accumulations of excrement refuse, and from pigsties. Byelaws not enforced, and no serious attempt made to deal with nuisances. Public water supply good.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
12. Holyhead and Llanddausaint Registration sub-districts.—*cont.*	Regis.-General's Returns	Holyhead rural sanitary authority.	*Holyhead Rural Sanitary District.*—Two outbreaks of diphtheria amongst a number of isolated farmhouses. In one outbreak channels of propagation of the disease not satisfactorily made out, but extension probably due to a school. In second outbreak, occurring in farmhouses subsisting almost entirely upon their own produce, disease shown to be mainly associated with attendance at a particular chapel. General condition of district very filthy.
13. Ince in Makerfield (Lancashire.) Population, 15,000. *Dr. Parsons.*	Reports of Medical Officer of Health.	Ince in Makerfield local board.	Persistent high mortality from all causes. Large prevalence and fatality of zymotic diseases. High infant mortality. Prevalent neglect and mismanagement of children. Large and offensive midden privies in general use, and system of scavenging inefficient. Sewer ventilation defective. Water supply good and wholesome. Private streets and courtyards not metalled, channelled, nor levelled, and in filthy condition. Great carelessness of the population with reference to the spread of infectious disease. No disinfection apparatus or provision for isolation of cases of infectious disease.
14. Newquay - - (Cornwall.) Population, 1,121. *Dr. Ballard.*	Information from Medical Officer of Health of an outbreak of enteric fever.	Newquay local board	Enteric fever of frequent occurrence. Recent serious outbreak in some new houses at Tolcarne Head, due to faults of drainage and to the use of a well water polluted with sewage. Considerable neglect of ordinary sanitary administration, and prevalence of nuisances.
15. Okehampton - - (Devon.) Population, 2,000. *Dr. Blaxall.*	Local complaints - -	Okehampton local board.	Water supply exposed to pollution by direct communication between water mains and closets. Sewerage unventilated, and very defective and imperfect. Privy accommodation insufficient and objectionable. Excremental nuisances general. Aggravated nuisances in connexion with pigstics, and accumulations of manure and refuse. No hospital provision for cases of infectious disease. Neglect of authority to properly carry out their duties.
16. Penistone - - (Yorks.) Population, 2,000. *Dr. Thorne Thorne.*	Report of Medical Officer of Health.	Penistone local board	Fatal prevalence of enteric fever and of scarlet fever. Water supply deficient in quantity and very generally polluted. Means of drainage most imperfect and the cause of considerable nuisance; midden privies a source of nuisance and of grave injury to health. No means of isolation or of efficient disinfection. General sanitary administration very unsatisfactory.
Pontypool Registration sub-district. (Monmouthshire.) Population, 25,287. *Dr. W. Ogle.*	Regis.-General's Returns		High general death-rate, and undue prevalence of certain zymotic diseases, notably scarlet fever. Prevalence of these diseases due to the close aggregation of the population in houses often ill-ventilated and overcrowded; to very general filthiness of bodies, clothing, and houses; to indifference of the working classes to ordinary sanitary precautions; and to neglect of the authorities to provide means of isolation, disinfection, or cleanliness.
	" "	Pontypool local board	*Pontypool Urban Sanitary District.*—Main sewer entirely unventilated, and in some places very imperfectly laid. Pans of waterclosets filthy, there being no provision for flushing other than by hand. In poorer neighbourhoods closet accommodation inadequate. Cottages substantially built, but filthy and badly ventilated. Scavenging fairly efficient. Slaughter-houses very filthy. Water supplied by waterworks to district good, but a considerable number of houses supplied by wells liable to dangerous pollution.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
17. Pontypool Registration District—*cont.*	" "	Panteg local board -	*Panteg Urban Sanitary District.*—At *Sebastopol* the pits of the common privies communicate with the sewers (which are unventilated) by an overflow pipe. Water supply from shallow wells liable to pollution. At *Cwmynyscoy*, sewer unventilated, and privies in connexion with it. Slops and filth thrown about anywhere. Water supply sufficient, but unnecessarily exposed to pollution.
	" "	Abersychan local board	*Abersychan Urban Sanitary District.*—Most of the villages composing the district sewered more or less completely, but sewers unventilated, and closets flushed only by hand. In those places where no sewer exists, closet accommodation is usually extremely bad. Scavenging fairly carried out, and slaughter-houses clean. Cottages dirty through unclean habits of occupiers. Structural sanitary works apparently suspended through depression of trade. Water supply in some of the outlying villages defective, and that supplied by a company thick, turbid, unfiltered, and unpleasant.
18. Quickmere - - (Yorkshire, W.R.) Population, 3,800. *Mr. John Spear.*	Regis.-General's Returns	Quickmere local board	Sustained high death-rate, and frequent outbreaks of zymotic disease. Drains, originally intended only for storm and surface water, imperfect and leaky. Excrement and house-refuse stored for lengthened periods in privy middens of the worst description. No system of public scavenging, and many nuisances of a grave character. House accommodation decent and substantial, but ventilation imperfect, and many houses built back to back. Water supply of township proper good, but that of outlying villages insufficient and liable to pollution. No hospital accommodation. Sanitary administration very defective.
19. Redhill and Caterham. (Surrey.) Population:—Caterham, 2,800. Redhill, 9,500. *Dr. Thorne.*	Local information -	Reigate town council and Godstone rural sanitary authority.	Extensive epidemic of enteric fever occurring simultaneously over the area supplied by the Caterham Waterworks Company, and limited to the consumers of the Company's water. Water ascertained to have been contaminated by means of the evacuations of a man employed in the construction of an adit between two of the Company's deep wells, whilst suffering from profuse diarrhœa attendant on an attack of unrecognised enteric fever. (*See* p. 78.)
20. Selborne - - (Hants.) Population, 697. *Dr. Blaxall.*	Request of Sanitary Authority.	Alton rural sanitary authority.	Outbreak of enteric fever following importation to the village of a case of that disease; subsequently spread by polluted water and infected privies. Water supply derived from wells which are much exposed to pollution. Filthy privies. Neglect of excrement removal. General want of drainage.
21. South Stoneham Rural Sanitary District. (Hants.) Population, 22,609. *Dr. Blaxall.*	Local complaints - -	South Stoneham rural sanitary authority.	High mortality from preventable diseases, and successive epidemics of scarlatina. Entire absence of efficient sewerage. Water supply mainly derived from wells greatly exposed to excremental pollution. Water from extraneous sources recently introduced, but hitherto little used by the public. Excrement disposed of in cesspit privies. General prevalence of aggravated excremental nuisances. Dairies situated in the midst of filthy surroundings. No hospital provision for infectious diseases, and no measure adopted to prevent the spread of infectious disease. Sanitary administration very defective.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
22. Stourbridge Registration District. (Worcestershire and Staffordshire.) Population, 73,386. *Dr. Parsons.*	Regis.-General's Returns and local complaint.		Extensive prevalence of scarlet fever amongst a manufacturing population. High general death-rate, and excessive mortality from preventable diseases. Scarlatina chiefly spread in recent epidemic by unchecked personal intercourse between the healthy and infected. Many insanitary conditions found prevailing in the district, especially accumulations of filthy water on surface of courtyards and in road channels, due to lack of proper sewerage. No means of isolation for the reception of infectious cases, and no disinfecting apparatus provided.
	" "	Stourbridge rural sanitary authority.	*Stourbridge Rural Sanitary District.*—Older cottages damp, ill-ventilated, and unfit for human habitation. Newer houses built without any supervision over their construction. Sewerage defective throughout the district, and many parts wholly unsewered. Private streets unrepaired and in a filthy and unwholesome condition. Water supply from wells and springs scanty and polluted. Public water-services laid down in most parts of the district, but not much used by inhabitants. Foul midden privies, of huge size and of bad construction. No public arrangements for removal of night-soil. Abatement of nuisances much neglected.
	" "	Quarry Bank local board.	*Quarry Bank Urban Sanitary District.*—No sewerage other than rough channels by roadsides. Water supply from wells liable to contamination. Foul, ill-contrived midden privies. General remissness in abatement of nuisances.
	" "	Stourbridge Improvement Commissioners.	*Stourbridge Urban Sanitary District.*—Some of the poorer houses miserably dilapidated, and in certain cases very dirty and overcrowded. Sewerage somewhat improved since Dr. Ballard's inspection in 1873, but still defective, and sewers unventilated. Water supply partly from wells liable to pollution. Foul, ill-constructed midden privies causing grave nuisance.
	" "	Brierley Hill local board.	*Brierley Hill Urban Sanitary District.*—Majority of streets sewered, but subsidence of ground through coal-workings necessitates continual attention to the sewerage. Wet open midden privies of large size. A considerable amount of sanitary improvement since former inspection in 1873.
23. Sutton Bridge (Lincolnshire.) Population, 2,081. *Dr. Parsons.*	Local complaints of neglect of local board.	Sutton Bridge local board.	Older dwellings ill-ventilated and very damp. Newer houses erected on a better plan, but some built with very improper materials. Unsatisfactory supervision of Authority over new buildings. Byelaws not properly enforced. Greater part of district entirely without sewerage. Dumb wells for disposal of liquid refuse, causing frequent nuisances. System of excrement disposal (by vault privies) the source of grave nuisance. Water supply from wells and rain-water cisterns greatly exposed to pollution. No hospital accommodation for infectious diseases, and no disinfecting apparatus.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
24. Swimbridge (Devon.) *Dr. W. Ogle.*	Regis.-General's Return	Barnstaple rural sanitary authority.	Epidemic of diphtheria in a circumscribed area within a limited space of time. Causation of outbreak doubtful, and cottages invaded had nothing in common as to water or milk supply. Village school apparently the effective agency in the dissemination of the infection.
25. Swindon Registration Sub-District. (Wiltshire.) Population (1871), 17,459. *Dr. Blaxall.*	Regis.-General's Returns		Extensive epidemic of scarlatina mainly spread by intercommunication of the infected and the healthy. School congregation an active agent in the propagation of the disease. General carelessness as to infection, and neglect of authorities to secure isolation or disinfection, or to enforce precautionary measures. Existing hospital for infectious cases not utilised for the reception of any of the cases.
	" "	New Swindon local board.	*New Swindon Urban Sanitary District.*—Great and rapid increase of population. Dwellings damp and thickly peopled. Incidence of measles mortality associated with dampness of dwellings. Excrement disposal by means of privies discharging into sewers, but unprovided with means of flushing. Many of the privies very neglected, and in certain parts of the district cesspit privies in use. Sewers defective and unventilated. Supply of water from waterworks intermittent. Scavenging and removal of house-refuse undertaken by authority, and apparently satisfactorily performed.
	" "	Old Swindon local board.	*Old Swindon Urban Sanitary District.*—House accommodation generally good, but certain cottages old and out of repair. Excrement disposal by closets discharging into sewers, but with no means of flushing other than by hand. Large and unwholesome cesspit privies found in various parts of district. Sewers faultily constructed and inefficiently ventilated. Sewage outfall offensive. Water supplied by reservoirs, by a spring, and by a few local wells. Refuse removal undertaken by authority, but unsystematically performed.
26. Trottescliffe (Kent.) *Dr. Ballard.*	Report of Medical Officer of Health.	Malling rural sanitary authority.	Eleven cases of diphtheria, seven of them fatal, in a single family of 14 persons, residing at a cottage upon some farm premises. Cottage adjoined a churchyard in a low situation, and was damp from the foundation. Faults of drainage found on premises, which were also overrun with rats and mice.
27. Wells Rural Sanitary District. (Somerset.) Population, 11,972. *Mr. T. C. Langdon.*	Reports of Medical Officer of Health.	Wells rural sanitary authority.	Water supply in some parts of the district much contaminated, and in others so circumstanced as to be liable to pollution from excremental and surface soakage. Means of sewerage and drainage very generally defective. Privies faulty in construction and the cause of considerable nuisance. No means for the isolation of infectious cases. Administration of the sanitary authority very imperfect.
28. Wickford (Essex.) Population, 491. *Dr. Thorne Thorne.*	Report of Medical Officer of Health.	Billericay rural sanitary authority.	Prevalence of diphtheria in connexion with conditions of faulty drainage and of excrement disposal causing grave nuisance. Wickford village frequently flooded, owing to low site and obstructions in the bed of the river Crouch.

Area of Inquiry and Name of Inspector.	Ground of Inquiry.	Jurisdictions Concerned.	Chief Facts reported by Inspector.
29. Wing - - (Bedfordshire.) Population, 1,520. *Dr. Blaxall.*	Local request - -	Leighton Buzzard rural sanitary authority.	Epidemic of enteric fever in a circumscribed area, associated with the use of water from a well obviously polluted by soakage from privies and drains. Excremental nuisances generally prevalent, and a source of danger to health. Neglect of authority to carry into effect the recommendations made by Dr. Buchanan in 1870.
30. Yeadon - - (Yorkshire, W.R.) Population, 5,700 *Mr. John Spear.*	Regis.-General's Returns and local complaints.	Yeadon local board -	Violent epidemic of scarlatina diffused throughout the district. Water (supplied by company) grossly polluted in course of collection. Hitherto no system of sewerage, but works now begun. System of excrement disposal (by privy middens) more than ordinarily offensive, and the cause of nuisances of the gravest kind. Many houses dilapidated and unfit for habitation. Back-to-back houses frequent. Lodging-houses unregulated and objectionable. No hospital accommodation for infectious cases. Sanitary administration lax.

No. 6.

On an Outbreak of "Fever" that proved to be of the nature of Trichiniasis on board the Reformatory School Ship "Cornwall," by Mr. W. H. Power.

This inquiry was directed by the Local Government Board at the request of Mr. Secretary Cross, to whom application had been made (30th October 1879) by the School Ship Society for investigation of an alleged outbreak of "fever" on board their ship "Cornwall."

This outbreak, it was stated, had occurred about a month before, and was the second prevalence of "fever" on board within a period of four years. At the end of 1875, indeed, nearly 50 per cent. of the boys had been affected with what was held to be enteric fever. In regard of this outbreak, and also at the request of Mr. Secretary Cross, an inquiry was in March 1877 ordered by the Local Government Board. It was to have reference to the sanitary condition of the "Cornwall," and to a suggestion that the health of the boys had suffered from the outflows of metropolitan sewage at Crossness and Barking. Mr. Netten Radcliffe conducted this inquiry, but for various reasons, was unable to complete it. A memorandum by him presented to the Board on the subject is dated May 1878.

The "Cornwall" lies at moorings in the Thames off Purfleet. Formerly, as an 80-gun ship, her complement consisted of 800 officers and men. As a reformatory school ship she has at no time accommodated more than 318, boys and officers. At the date of the recent outbreak the numbers were 262 boys, and 15 officers. The age of the boys ranged from 12 to 20 years.

The outbreak of 1879.—Up to mid-September the health of the boys had during a long period been satisfactory. For many weeks no boy, except one accredited with incipient phthisis, had been more than a very few days on the sick list, and the entries appearing on this list referred only to minor ailments and to casualties. On *Tuesday*, 23rd September, seven boys reported themselves to the medical officer of the ship for the sick list and were admitted to the sick-bay. Setting aside one trivial case, six boys suffered thenceforward from an illness that lasted 18 days in a fatal case, and 26, 37, 38, 42, and 88 days in cases that recovered. Next day, 24th September, two boys reported themselves to the doctor; one of them was only slightly ill for a few days, the other was confined to bed 28 days. By the 29th of September 10 other boys had come on the sick list. Except one who had symptoms of pneumonia, and was ill 38 days, none had marked objective symptoms, and all of them seemed well in a few days. On *Tuesday*, 30th September, and *Wednesday*, 1st October, six boys came under treatment. Three of them had illness lasting 18, 18, and 29 days; the ailments of the others appeared trivial. Between 3rd and 6th October nine boys, and on 11th October one boy came under the doctor. All, except one who suffered several weeks from jaundice, were out of sorts a few days only. From *Tuesday* 14th, to Thursday, 17th October, six boys came under treatment. Five of them were ill for 18, 18, 31, 35, and 67 days respectively; one only proved a trivial case. On Tuesday, 21st October, one boy, and on 23rd October another boy came under treatment. The former was ill 14 days, the latter had no decided symptoms. This was the last case up to the time of the inquiry. In all, therefore, there were 43 cases of some sort or

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degree of sickness. They may conveniently be divided into the more serious and the less serious attacks. In all 18 boys suffered from decided, most of them from serious, illness, and one of them died on the 18th day of his disease. Twenty-five other boys were slightly ill, most of them for a few days only; and some of these 25 may not, it was thought, have been ill at all, but owing to panic may have imagined themselves possessed of symptoms of the sort described by boys who really suffered. There can be no doubt that all the serious cases, and some of the slighter ones as well, had a disease which was properly described as a "continued fever." In most instances the symptoms were clearly indicative of enteric disturbance, and in some cases there were in addition persistent diarrhœa, rose-coloured spots on the abdomen, and hæmorrhage to a greater or less extent from the bowels. From these data the outbreak was regarded as one of enteric fever, presenting perhaps an unusual number of abortive cases, and as possibly complicated by malingering.

On occurrence of the outbreak prompt measures were taken for isolation of the boys attacked. In the absence of any proper hospital accommodation in connexion with the ship, the boat-house at Purfleet, belonging to the vessel and situated on the jetty by which access is gained thereto, was at once cleared of its contents and prepared for reception of the sick. By 26th September it was fitted as a temporary hospital, and on that day seven cases were transferred to it. Subsequently, cases as they arose, whether severe or trivial, were removed to this boat-house. On 1st October, arrangements having been made for their reception, 10 boys were removed to the Seamen's Hospital, Greenwich, and on 6th October another batch of four boys. With the single exception of a case of jaundice, none of the boys removed to Greenwich had any notable illness; consequently 13 of the 14 were returned to the "Cornwall" after a week or 10 days each in hospital. During the progress of the outbreak a plot of ground at Wennington, 1½ miles from the ship, was acquired by the School Ship Society for hospital purposes, and hereon was erected an iron building to which were removed on 7th November all patients from the boat-house capable of bearing the journey. On 11th November, the occasion of my first visit to the "Cornwall," one boy only remained in the boat-house. This boy had then been ill 50 days, and presented, on such examination as was bearable by him under the circumstances, conditions usual to persons who have suffered many weeks from fever.

This was the history as presented to me at the beginning of my inquiry; and I at once set myself to search for causes of enteric fever.

In seeking for such causes regard was had to circumstances of a kind that experience has suggested might be concerned with enteric fever, and that may have affected (*a*) the ship's company collectively, or (*b*) particular boys, or groups of boys, and through them perhaps other persons. It was seen that if such circumstances failed to explain the occurrence of the disease, inquiry would have to be directed into the influence of articles of food hitherto not recognised as capable of producing enteric fever.

I. *a.* Under the first head the evidence obtained proved negative. *Water* could be excluded for the reason that the water consumed on board was from the Purfleet reservoir of the South Essex Water Company, within the area of which service no prevalence of enteric fever was heard of. Such risk of contamination of this water as was formerly incurred during transit to the ship has been got rid of since the outbreak of 1875 by

establishing, by means of a hose attached to a chain cable, direct communication between the mains of the water company and the water tanks on board: to this new method of water conveyance no suspicion attached. *Milk* as an agent of infection was altogether out of question. None of the boys on board under any circumstances partook of milk. *Sanitary Circumstances of the Ship.*—Herein nothing was discoverable suggestive of conditions likely to favour recrudescence of the disease of 1875, or to foster recently imported infection. Most of the structural and other alterations recommended after the last outbreak by the Port Medical Officer had been long since adopted, and the ship throughout appeared to be clean, well ventilated, and wholesome. Notably the arrangements and regulations respecting excrement disposal were, especially as regards the boys, of such sort as to render highly improbable any fostering or dissemination of infection of excremental outcome. It is true that two officers' closets on the main deck forward (which have been retained notwithstanding the Port Medical Officer's recommendation for their removal) are from their proximity to the sick-bay objectionable; but no evidence was forthcoming suggestive of their having had concern with the outbreak. *River Circumstances.*—In regard of possible influence of river-borne sewage, bathing and swimming of the ship's company in the river, and washing of decks with river water, received attention. As to bathing, all hands bathed for the last time for the season on the 15th August. Only boys learning to swim, some 24 in number, continued their lessons until the 8th September. Bathing seemed to be excluded as a factor in the outbreak from the circumstance that the interval between the occasion of the last bathing and the date of the outbreak, a period of upwards of five weeks, far exceeded any known limit of the incubation period of enteric fever. On the other hand swimming, though not thus excluded, was set aside from the fact that one only of the 24 boys learning to swim was included in the first group of sufferers by the outbreak. Washing, or rather sluicing of decks with river water could not, as a possible cause of the outbreak, be excluded: but on the other hand, there was nothing whatever to show that it had, in fact, any influence for harm. And so with any emanations there may have been from the river itself, or from the foreshore.

I. *b.* The second stage of inquiry, having to do with the circumstances of particular individuals and groups of individuals, required, beside other information more or less easily attainable, minute and accurate knowledge of the inner life on board the vessel. Thus it came about that I attempted to live over again, as it were, on shipboard the weeks preceding the outbreak. In doing this I was assisted, in a way and to an extent which I cannot describe, by the Captain Superintendent, who throughout a tedious inquiry rendered me unwearied assistance. By this method a mass of evidence concerning the boys was got together. It had to do with boys who escaped as well as with boys attacked, and much of it, as was inevitable, proved in the end irrelevant. Before proceeding to deal with the matters investigated it will be convenient to state in a tabular form, for future reference, some of the data obtained in regard of the boys attacked.

Date of going on Sick List.	Name and Age.	Date of Removal from Ship.	Duration of Illness to Convalescence. (Days.)	Watch.	Mess.	Division.	Position of Sleeping-berth.	Last on Leave before Outbreak.
September 23	Harry Sims, 15	September 26	88	Starboard	15	13th	Lower deck, right forward	No leave.
" 23	Henry Wheeler, 16	September 26	37	Port	17	16th	" " abaft main hatch	"
" 23	Joseph Bradshaw, 16	September 26	38	Port	3	4th	Middle " abreast main hatch	"
" 23	Arthur Brawnis, 16	September 26	26	Starboard	15	11th	Main " forward, ship's side	"
" 23	Charles Beaumont, 15	September 25	42	Starboard	15	5th	Lower " abaft main hatch	July 22–25.
" 23	Richard Pierce, 16	September 26	18—died	Starboard	15	13th	" " right forward	No leave.
" 23	*Alfred Tuck, 16*	*October 1*	*10?*	*Port*	*17*	*18th*	*Lower " forward, ship's side*	*August 2.*
" 24	Edward Davis, 16	September 26	28	Port	3	4th	Middle " abreast main hatch	September 26, 1878.
" 24	*Henry Dashwood, 16*	*October 1*	*8?*	*Port*	*6*	*10th*	*Lower " abreast hatch, forward*	*April 12.*
" 26	*George Brace, 16*	*October 1*	*5?*	*Starboard*	*6*	*9th*	*" " opposite magazine hatch*	*May 27.*
" 27	*George Muir, 15*	*October 3*	*6?*	*Port*	*5*	*6th*	*Middle " abreast fore-ladder*	*April 10.*
" 28	Arthur Marks, 16	On board throughout	38	Starboard	19	17th	Lower " abaft main hatch	December 27, 1878.
" *28*	*James Austen, 16*	*October 1*	*?*	*Starboard*	*3*	*3rd*	*Middle " abreast model*	*No leave.*
" *29*	*Alfred Jardine, 15*	*October 1*	*?*	*Starboard*	*9*	*9th*	*Lower " " hatch forward*	*June 20.*
" *29*	*Alfred Mortby, 14*	*October 1*	*?*	*Port*	*9*	*13th*	*" " forward*	*June 8.*
" *29*	*Edward Dowsett, 15*	*October 1*	*?*	*Port*	*6*	*10th*	*" " abreast hatch, forward*	*April 26.*
" *29*	*John Loader, 13*	*October 1*	*?*	*Port*	*10*	*14th*	*" " amidship*	*May 6.*
" *29*	*Joseph Childs, 16*	*October 1*	*?*	*Starboard*	*10*	*7th*	*" " opposite main hatch*	*No leave.*
" *29*	*Henry Rose, 15*	*October 1*	*?*	*Port*	*5*	*4th*	*Middle " " " "	*May 18.*
" *30*	*William O'Hara, 16*	*September 30*	*?*	*Port*	*3*	*4th*	*" " " "	*August 28.*
" *30*	*George Saunders, 15*	*October 1*	*?*	*Port*	*19*	*18th*	*Lower " abaft cabins*	*No leave.*
" *30*	*James Kiff, 16*	*September 30*	*?*	*Port*	*8*	*10th*	*" " opposite magazine*	"
October 1	Henry Salmon, 16	October 3	18	Port	11	8th	" " opposite main hatch	September 14–18.
" 1	Charles Speer, 17	October 2	18	Port	4	4th	Middle " " "	" 7–11.
" 1	William Putt, 15	October 2	29	Starboard	15	9th	Lower " " magazine hatch	April 16.
" *3*	*George Lawrence, 15*	*October 3*	*?*	*Starboard*	*8*	*5th*	*" " abreast main hatch*	*No leave.*
" *3*	*W. Spice, 15*	*October 3*	*?*	*Port*	*20*	*18th*	*" " abaft cabins*	"
" *3*	*J. Mitchell, 15*	*October 3*	*?*	*Port*	*20*	*18th*	*" " " "	"
" *3*	*George Newman, 17*	*October 3*	*?*	*Starboard*	*3*	*1st*	*Middle " abaft "	*June 3.*
" *5*	*Alfred Ings, 15*	*October 9*	*?*	*Port*	*20*	*18th*	*Lower " abaft cabins	*No leave.*
" 6	Frederick Simmonds, 16	October 6	38	Starboard	5	5th	" " opposite main hatch	May 28.
" *6*	*William Moseley, 16*	*October 16*	*?*	*Starboard*	*1*	*1st*	*Middle " abaft cabins	*July 3.*
" *6*	*Albert Ferris, 16*	*October 16*	*?*	*Port*	*1*	*2nd*	*" " " "	*May 31.*
" *6*	*Daniel Holland, 15*	*October 15*	*?*	*Port*	*11*	*12th*	*Lower " forward	*April 19.*
" *11*	*Henry Rogers, 14*	*October 11*	*8*	*Port*	*4*	*8th*	*" " abreast officer's cabins*	*May 28.*
" 14	Edwin Brice, 16	October 15	31	Port	3	10th	" " opposite magazine hatch	May 31.
" 14	James Howes, 14	October 15	35	Starboard	17	15th	" " under main hatch	July 4–9.
" 14	Henry Weare, 15	October 14	67	Starboard	9	9th	" " opposite magazine hatch	May 30.
" *16*	*William Brooks, 14*	*October 16*	*?*	*Starboard*	*18*	*1st*	*Middle " abaft	*July 25.*
" 17	Frank Bowler, 15	October 17	18	Port	8	16th	Lower " under main hatch	No leave.
" 17	James Hopcroft, 15	October 19	18	Port	19	18th	" " abaft "	"
" 21	George May, 15	October 21	14	Port	17	12th	" " forward	August 4–6.
" *23*	*Frederick Ringrose, 14*	*October 24*	*8*	*Starboard*	*7*	*7th*	*" " opposite main hatch	*No leave.*

The boys on board the "Cornwall" are divided primarily into two watches, port and starboard, of equal strength; and are also classed in messes which are 20 in number in all. A third sort of grouping of the boys, in 18 "divisions," has reference to seniority and to sleeping quarters. The watches, each on alternate days, are employed on the work of the ship, seamanship-instruction, &c.; the watch not on duty being at school. On one afternoon in each week, Wednesday, all hands go ashore for recreation—cricket, football, and the like. The messes, each numbering 12 or 13 boys, have reference only to meals, and all meals are taken on the middle deck. Except at meal times boys of the same mess do not necessarily associate together. The sleeping berths, which are allotted without reference either to watches or messes, are with few exceptions on the middle and lower decks. Inmates of the sick-bay and wet-a-beds alone sleep on the main deck.

Data of the sort furnished by the table were studied with reference to the domestic life on board the ship here glanced at, but they did not for a length of time afford any clue to the outbreak. Of the many directions in which inquiry was pushed without result, some of the chief are as follows:—

Nothing could be discovered leading to suspicion that any groups, large or small, of boys could have been infected while *on shore* for amusement or exercise; and, beyond a somewhat heavy incidence of the early outbreak on a particular mess (an incidence that could not it was thought be usefully considered until other questions had been exhausted), no sort of grouping led to any suggestion about the origin of the disease *on the ship*.

Importation of infection to the ship by any individual boy seemed to be excluded. None of the boys early or severely attacked had been from the ship for many weeks previous to the outbreak. As regards new comers, in July seven, in August four, and in September two boys were admitted to the ship. One only of these 13 came under the doctor within two weeks of admission. He had slight diarrhœa on July 7th, 8th, and 9th. Short "leave" to boys who have been six months on board is often accorded, but only on written assurance by the friends to whom the visit is to be paid that their households are free from sickness. In the 12 weeks ending 23rd September 78 boys had leave, in no case exceeding five days. One only of the 78 came on the sick list within two weeks of his return. He suffered from "stomach derangement and debility" on July 24th, 25th, and 26th, after which he returned to duty.

Importation of infection by visitors was considered. Visitors to the ship, notably "old boys," are very numerous. In the month of August alone, 42 old boys (14 of them on one day, 21st August) visited the ship. Old boys and other male visitors use (if they require to do so) the closets in the "heads"; females use the private closets. It has been already noted that spread of infection by means of the closets on board appeared altogether unlikely. Inquiry is made of all visitors to the boys as to infection at their homes before they are received on board.

In the laundry arrangements no suggestion as regards importation or dissemination of the disease was afforded. All clothing of the boys is washed on board, in water from the domestic supply, on Mondays and Thursdays, and each boy does his own washing. Clothing and bed linen of sick cases were during the outbreak disinfected at the boat-house and then taken to Purfleet to be washed.

Particulars respecting the dates of reception on board of new clothing and bedding, and the distribution of such articles to boys individually or in groups, afforded no clue to the outbreak.

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II. It was not until the foregoing lines of inquiry had been exhausted, that inquiry as to possible influence of food in producing the outbreak could be usefully made: for we have no knowledge of dissemination of enteric fever by constituents of food other than water and milk. In connexion, however, with the question of food of the boys, certain circumstances that had throughout the inquiry been borne in mind began to attain prominence. These were, the fact that the food supplied to and cooked for the boys was distinct and separate in all respects from that of the officers, who escaped illness; that the incidence of the first outburst of disease had been exceptionally on mess 15; and, that the more serious cases arranged themselves in groups which were attacked mainly on *Tuesdays* or *Wednesdays* at weekly or fortnightly intervals. Thus in seeking among articles of food for a heretofore-unrecognised agent in the propagation of enteric fever, there existed no primâ facie ground for suspecting one kind of food more than another, unless one could be found capable of fulfilling certain assumed conditions of the problem requiring solution. These conditions were, that the article of food should have come into use about the time of the outbreak; that it should have been consumed by the boys alone, and chiefly in the first instance by boys of a particular mess or messes; and that it should have had opportunity of operating weekly for two successive weeks, and then only after the lapse of a fortnight. Considerable hesitation was felt as to the validity of the conditions assumed as requiring fulfilment. For the heavier incidence of the outburst on mess 15 was not so decided as to be beyond the range of mere chance, and the assumption of operation of the cause of the outbreak on certain Tuesdays was based on one class of cases only, the graver ones, as probably being in their incubation more constant than the others. Obviously, too, all articles of food provided solely for the boys would equally fall under suspicion, if the fact of boys alone being attacked were exclusively considered. But notwithstanding objection to seeking hypothesis to fit hypothesis, investigation in the above sense was undertaken, for the reason that unless this was done inquiry could not proceed further.

Exceptional food of the boys first received attention, and herein was found a circumstance that seemed of possible significance. Pierce, the boy who died, received, as boys were in the habit of doing, a present of food from home, shortly before his illness. This food consisted, it was thought, of cake alone, which he probably shared with his mess (No. 15), and other comrades. By this means might have been brought about (if the cake was infective) some of the earlier features of the outbreak; though all the conditions of the problem would not thus be satisfied. The question was therefore left for, if necessary, future consideration.* Inquiry respecting the regular diet of the boys extended to all articles of food used on board, the days of the week on which they were habitually consumed, and the dates on which articles of food other than fresh meat and vegetables (which were received day by day) were received on board. As regards meals, breakfast and supper offered no hint as to the cause of the outbreak. Neither meal varied in any way suggestive of relation with the grouping of cases observed in the outbreak. Dinner, however, seemed to suggest a clue. The animal food of the boys, though varied day by day (fresh meat four times a week, and cheese, suet pudding, and salt meat each once), was the same every Sunday, every Tuesday, every Wednesday, every Thursday, every

* Toward the close of this inquiry, and after the above was written, the Captain Superintendent ascertained that Pierce's present comprised, besides cake, in all probability animal food. The further consideration of the subject which thus became necessary will be given later on (page 57).

Friday, and every Saturday; but on *Mondays* could vary. Monday was, indeed, always a boiled *salt meat day;* but the *sort* of salt meat consumed on Mondays differed from week to week. As a rule it was alternately salt beef and salt pork; now and then it happened that either one or the other constituted dinner on *successive* Mondays; and occasionally a dinner of the one was supplemented to some extent by the other. Salt beef, therefore, and salt pork came under suspicion, for either of them might by this method of consumption have been related to the grouping of the graver cases on or about certain Tuesdays; and further, it was ascertained that a fresh cask of each came into use shortly before the outbreak. Suspicion of this sort once aroused settled naturally on pork. Not indeed on account of so-called typhoid fever of the pig (which is not known to be transferable to man), but for a wholly different reason. It occurred to me that if pork had had to do with the outbreak, some of that (224 lbs.) of the cask newly taken into use *might have been trichinised, and that the disease, therefore, from which the boys suffered might not have been enteric fever at all, but trichiniasis.* It will be observed that a theory of trichiniasis seemed exactly to fit the conditions assumed as requiring fulfilment. A newly broached cask of pork came into use shortly before the occurrence of the outbreak. The pork was consumed by the boys alone. Portions of the pork not thoroughly well cooked might form but a small proportion of a total dinner for the boys, and might therefore have been consumed on particular days within the limits of one or two messes, and thus (if such portions happened to be infected with trichina) the incidence of the severer cases on a particular mess or messes might have been brought about. Again, pork might very well have been consumed on two successive Mondays, and after that not until a fourth Monday, and thus the specialties of the time-distribution of cases, notably the peculiar grouping of the graver attacks, might be accounted for; while the great diversity of duration of illness would be quite intelligible. Trichinosis, in its incubation period, and in the duration of the illness resulting from it, has been known in outbreaks of the disease upon the Continent to vary very greatly. Probably the persons who have eaten most largely of trichinised flesh, or who have eaten it in a condition most nearly approaching a raw state, suffer soonest (sometimes within 24 hours) and most severely from trichinous disease. Many persons who have certainly partaken of trichinised flesh, but who have done so sparingly, or in a fairly cooked state, have escaped the disease altogether, or have had merely trifling illness of undefined commencement and end, and occurring many days, even some weeks, after the infective meal. Other evidence, too, seemed to support the theory. It was now ascertained that the use of pork on board the "Cornwall" had, owing to misgiving as to the wholesomeness of such food, been discontinued shortly before the cessation of the outbreak; further, it was learned that the pork in question was "American pork," exceptionally obtained from a firm in Bristol; and more, that it consisted solely of "belly pieces," and therefore of parts of many pigs, and of those parts most likely to be infested with trichina.

The theory of trichiniasis was accordingly put to the test of further inquiry; but no confirmation of it was at first to be found. Inquiry (1) respecting the actual consumption on particular Mondays of the pork in question, (2) the pathological condition of that pork, and (3) the clinical records of the cases attacked by illness failed, for various reasons, in attaining any other than a negative result. (1.) As to pork the cook's books, though showing day by day the consumption in kind and in amount of each article of food used by the boys, failed to distinguish

between salt beef and salt pork. These were habitually classed together as "salt meat," 96 lbs. of which were entered as having been consumed each Monday in September and October. Thus the precise Mondays in these months on which pork had been consumed, and that Monday in October on which it had been last eaten, could not be determined. Again, (2) microscopical examination of small portions, in each instance, of pieces of pork remaining in the "harness cask,"* and believed on board to have formed part of the contents of the cask that had come into question, failed to find any trichinæ. (3.) Detailed examination of the clinical records of the cases of illness failed in like manner to establish or to refute the theory. Few notes, except those relating to temperature, pulse, and respiration, had, owing to stress of work during the outbreak, been taken, and these by themselves afforded no ground for a conclusion. The temperature curves (when I had formulated the notes in charts) showed that beyond doubt many of the boys had suffered from continued fever; but as to the precise nature of that fever they gave no definite information. In several instances, and notably in their inception, these curves differed considerably from the temperature curves of enteric fever; but how far they (or any of them) corresponded with the temperature curves of fever resulting from trichinosis I had no sufficient means of judging. The pulse records of the cases did not in their relation to the temperature curves appear altogether suggestive of enteric fever.

It will be observed that this study of the clinical records of the cases, though it failed to elicit information directly suggestive of trichiniasis, failed to a like degree to support a theory of enteric fever. Attempt was therefore now made, in conjunction with the medical officer of the ship, to recover by inquiry of the boys themselves and of their attendants more extended particulars of the clinical history of the cases attacked. By this means, and with help of leading questions, it was ascertained that if the outbreak had been one of trichinosis, certain symptoms, usually described as characteristic of that disease, had not been prominent. Notably "sudden swelling of the face, particularly of the eyelids, at an early stage of illness" had not, except perhaps in one or two instances, been noticed.† No obvious "painfulness and immobility of the arms and legs with œdema and contraction of the muscles" could, except doubtfully in a single instance, be heard of. Again "œdema of the feet, legs, and thighs toward a later stage of illness" had not, except in one case, existed. General "anasarca of the trunk" had been altogether absent. But nevertheless it became apparent that though in no single instance had all the symptoms referable to trichiniasis been present, still in several cases one or more symptoms that might have belonged to that disease had existed, and in other cases such symptoms seemed obscurely hinted at. Further, in some half dozen instances, there had been jaundice of an anomalous sort. Upon the whole it did not seem that trichiniasis could, from this clinical history, be set aside; though evidence affirmative of it was, thus far, wanting. On the other hand, these new details left me unconvinced that the outbreak had been enteric fever; and this, notwithstanding the occurrence in certain cases, of symptoms more or less diagnostic of that disease. As the questioning of the boys and of the ship's staff proceeded, it became,

* The "harness cask" is that containing brine, into which odd portions of the salt meat taken from the original casks await consumption. The amount of meat that finds its way into the harness cask probably varies a good deal.

† The symptoms here put in inverted commas are quoted from Dr. Thudichum's account of the diagnostic points of the disease.—(Seventh Report of Medical Officer of Privy Council, pp. 379–80.)

indeed, increasingly evident that a large proportion of the fever cases might be more safely ranged under some general name, such as "simple continued fever," than under the specific name of enteric fever; and it was observed, too, of the less febrile cases that they, like the more febrile, were accompanied by pain in the belly and nausea, with, though commonly without, looseness of the bowels.—In taking this view of the outbreak, I found myself in much accord with the Medical Officer of the ship, who, except for some few cases, would from the beginning have been disposed to avoid a name that implied a certain knowledge of the pathology of the disease.

It was now, upon reaching this point, that it came to be pretty clearly seen that an outbreak, characterised as this one was in the bulk of its cases, by "simple continued fever" with gastero-enteric disturbance, might just as well have been related to trichiniasis as to enteric fever; the fact of certain of the graver cases of the outbreak, taken collectively, presenting most of the symptoms characteristic of enteric fever, seeming to lose significance beside the circumstance that these very same cases presented, collectively, many symptoms that might be referable to trichiniasis. So that, at this stage of inquiry it had become obvious that need for *direct* evidence respecting the nature of the outbreak had become imperative, and in this sense I reported to the Medical Officer of the Board.

Two methods were indicated whereby direct evidence might be brought to bear on the subject of inquiry. One, microscopical search for trichinæ in portions of muscle excised from patients who had recovered; the other, exhumation and pathological examination of the body of the single fatal case of the outbreak. The former course did not commend itself; for microscopical examination of living muscle is practicable only within somewhat narrow limits, and failure to find trichinæ by this method, while not necessarily disproving trichiniasis, would in no sense afford evidence of enteric fever. On the other hand exhumation and pathological examination of the body (with the large facilities thus procured for microscopic examination of muscles) of the boy who died might be trusted (if decomposition had not too far advanced) to decide between trichiniasis and enteric fever. Further, this course had the additional advantage of dealing with what was in effect a test case of the outbreak. The case in question was that of the boy Pierce, who was one of the first group of sufferers attacked on September 23rd. He in particular exhibited symptoms (persistent diarrhœa, copious eruption of rose-spots, and even free hæmorrhage from the bowels) believed at the time to be those of enteric fever. In addition, he, in dying on the 18th day of his illness, appeared to be the subject of bowel perforation.

Application was therefore made by the Board to the Home Office for an order for exhumation of the body of Pierce. An order being granted conditionally on consent of the friends of the boy being first obtained, and on the understanding that the exhumation and necessary re-interment of the body should be carried out under the auspices of the Sanitary Authority of the district in which burial had occurred, I set myself to procure this consent and to make the requisite arrangements. This having been done, the examination of the body took place on the 6th December. With the consent of the Board, Dr. Cory (who had previously assisted in microscopic examination of the pork) was associated with me at the necropsy and in the pathological investigations which followed.

It was hardly expected that post-mortem examination of the ordinary kind would, two months after burial, afford much information. It was

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thought that detailed pathological examination of certain viscera and tissues would be needed before a conclusion could be arrived at. At the necropsy, however, it was found that decay had not so far advanced as to obscure certain first indications which, while they did not point to trichiniasis, went far to negative the notion of enteric fever. The body, though much decomposed externally, fairly retained its normal proportions. There was no œdema of the feet, limbs, or trunk. Internally the effects of decomposition were less manifest; the various viscera were easily made out, and so far as naked-eye appearances went, were not much changed. Beyond enlargement of the spleen, which was double the normal size, there was nothing suggestive of enteric fever. There was no peritonitis, nor any evidence of perforation. The peritoneum generally was clear, shining, and free from lymph or adhesions. No enlargement of the mesenteric glands could be discovered. The stomach, intestines, some of the other viscera, and portions of various muscles were reserved for further and more exact examination.

Further examination was carried out conjointly by Dr. Cory and myself. I think it right to put on record minute technical details respecting this examination, for the use of future inquirers. First, as to enteric fever. Very careful examination of the stomach and intestines failed to find any trace of ulceration, or (allowing for decomposition) of any change in the intestinal glands such as is usual in enteric fever. Moreover, the bowels, as tested by water, proved throughout intact. In a few places the bowel was discoloured internally, as if by injection of the mucous membrane subsequently altered by decomposition; but there was no appearance among the decomposed bowel contents of the presence of blood in any considerable quantity. More thorough examination of the mesenteric glands than had before been practicable revealed a few only enlarged, and none were bigger than peas. The other organs afforded no evidence for or against enteric fever. It thus appeared, after detailed examination of the viscera habitually implicated in enteric fever, as had appeared from the original necropsy, that with all allowance for decomposition, no such anatomical changes as are characteristic of enteric fever toward the end of the third week of its duration were to be found; and the conclusion now seemed warranted that whatever had been the nature of this boy's fatal illness, it certainly had not been enteric fever.

See App. A. and subjoined Report by Dr Bastian.

This conclusion was more than confirmed by the results of microscopic investigation, conducted with reference to trichiniasis. In the very first specimen examined, a few fibres from one of the abdominal muscles, was found a *wandering and living trichina;* and further search revealed the presence of these parasites in most of the muscles examined.* Although tolerably abundant, the trichinæ could not be said to "infest" (in the common sense of the word) any of the muscles, except perhaps the diaphragm, and in none of them had the parasite reached the stage of encapsulation. Except the first found, no trichinæ exhibited active movement, and comparatively few had attained, as had that one, their full growth as muscular parasites. In specimens of this sort the internal structure and organization were under a high power ($\frac{1}{12}$) readily made out, the parasites having altogether resisted decomposition, and most of them having probably died only a short time before examination. The

* No trichinæ were discovered in the heart. This organ was found to be small, its walls thin and flabby, and its muscles more decomposed than any examined. Possibly during life the heart had been severely trichinised (seeing that brood parasites, if conveyed by the venous blood, would encounter it first of all the muscles) though subsequently the parasites had migrated from it. Encapsuled trichinæ in the cardiac muscles are almost unknown.

smaller, not fully grown, muscular trichinæ, on the other hand, had seemingly all long since died. Their outline had become indistinct, their interior granular, and their structure could not be made out. Many were found broken up. Examination of the decomposing contents of the small intestine failed to find any parent trichinæ, but by scraping the under surface of the folds of mucous membrane of the bowel, brood trichinæ in considerable numbers were obtained. These had apparently all died, and their structure could not satisfactorily be made out.

Doubt could no longer exist that this boy died from acute trichiniasis. Not only was the presence in his body of the actual parasite demonstrated, but the results of microscopic examination pointed to the parasites as having played a very active part in the production of his illness, and were indeed precisely such as might have been anticipated in a case of early death from trichina disease. Death took place in this instance on the 18th day after appearance of symptoms, at a period that is too short to have allowed of universal migration of brood trichinæ, and insufficient also for the attainment of full growth in the muscles of any but early migrants. At the same time the period was not nearly long enough to have sufficed for the full evolution and encapsulation of many (if, indeed, of any) muscular parasites.*

Demonstration such as this of the nature of the fatal illness in what has above been termed a test case of the outbreak, was in effect demonstration of the nature of the outbreak itself. It will be remembered that the evidence adduced indicated that the cause (whatever that might have been) which produced this boy's illness, produced also the illness (so far as that was specific) of his shipmates; that his case, in that it formed one of the first and most severely attacked group of sufferers taken ill on 23rd September, was representative of the earliest and intensest operation of that cause; and that it was representative also, and in a high degree, not of the anomalous but of the *enteric* class of cases; of those, namely, whereon diagnosis of the outbreak had been in the first instance based, and in regard of which a theory of trichiniasis had appeared at one time well nigh untenable. Under the circumstances it was deemed unnecessary to proceed by vivisection of muscles of recovered cases to seek further and corroborative evidence as to the nature of the outbreak.

There now remain to be considered the circumstances under which trichina disease became conveyed to the boys. In this connexion must be noted a suggestion, made for the first time while this report is in progress, that *the ship's* pork had not been the *source* whence the trichinæ had been derived. It was ascertained by the Captain Superintendent that Pierce, the boy who died, had, among other things, in the present of food received from home shortly before his illness, a piece of fresh roast pork. This present of food (at the time thought to have been cake) has been already referred to on p. 52 of this report, where it is stated that although all the features of the outbreak could not be accounted for on the assumption that the food brought to Pierce was infective, still the illness of Pierce and his mess (No. 15), and other comrades attacked early in the outbreak, might possibly be so explained. Accordingly inquiry was made concerning this present of pork, with a

* It is here assumed that migration to and growth and development of the trichinæ within the muscles practically ceased with the death of their host. The circumstance that any living trichinæ should have been found in this boy's muscles two months after burial proves merely the extraordinary vitality of the parasite. But on these aspects of the subject, see Appendix A. to this Report.

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view of ascertaining what concern it might have had in the commencement of the outbreak. Considerable doubt existed in the first place as to the date on which this present of food was received; the evidence of some (and perhaps the most trustworthy) witnesses, fixing it at the end of August (three weeks to a month before the outbreak), that of others placing it at a much earlier period. The mother herself, who once only, she says, made her son a present of pork, thought she had done so shortly before the Bank holiday in August. Accounts, too, varied as to the amount of pork constituting the present; but upon this point perhaps the mother's statement is to be relied on. It was to the effect that she purchased, at 8*d.* per lb., 1¼ lbs. of loin of pork on a Saturday, and after dining on it with her daughter on Sunday, took the remainder to her son on visiting him on the following day. Neither she nor her daughter suffered subsequently from any illness. The pork was, she stated, well cooked, for the reason that she had an especial distaste for under-done pork. Of other witnesses on this point, some confirmed while others dissented from this statement. On one point only of the subject were the various witnesses unanimous: whatever may have been the quantity of the pork, the date of its reception, and the amount of cooking it had undergone, all agreed that Pierce consumed most of it himself. Two boys only could be found who acknowledged having tasted the pork, and neither of them was included in the list of sufferers by the outbreak. The members of Pierce's mess (No. 15), who were especially questioned on this subject, one and all denied having tasted the pork, and few of them were aware that Pierce had had any. It thus appeared that of the first sufferers by the outbreak, none excepting Pierce had partaken of the particular pork in question; and that of those, including the mother and sister, who had consumed that pork, none, again excepting Pierce, had suffered by the outbreak. Probably, therefore, this pork had no concern whatever in causing illness on board the "Cornwall."

The foregoing alternative having been disposed of, it seemed inevitable that the "American pork" on board the "Cornwall" was the source whence the trichina disease had been derived; and an attempt was now made to learn how it had happened that the method of cooking salt meat on board the vessel had failed to destroy the infectivity of the pork; and incidentally evidence was sought as to the extent to which the implicated pork (consisting of pieces from various animals, in two or more casks) might have been affected with trichina disease. Cooking salt meat on board the "Cornwall" is effected by boiling it 3½ to 4 hours; a process that seemed at first sight not unlikely to secure cooking of the meat, and one which has, so far as I could learn, commonly to a great extent achieved its object—the meat being spoken of as stripping easily from the bone. But when the details of the process come to be considered, cause appears for questioning the uniformity of cooking obtained by this method. Salt meat is cooked on board *in bulk:* 96 lbs. of beef or pork, as the case may be, are packed in a covered galvanised iron vessel of size just sufficing for the meat and the necessary water; the latter, when the meat is added to it, being nominally at boiling point.* The iron vessel with these contents is then placed over the central fire area of a stove of the "kitchener" type; and is assumed to be retained there from 8.30 a.m. until 12.30. Once only, it is said, during that period is the vessel interfered with for the purpose of adding more

* Plunging meat into water that is (or is nearly) boiling has been proved by experiment to retard cooking of the interior of the meat.

water.* Now, it will not be disputed that meat thus cooked on a large scale and in bulk cannot, except after very prolonged boiling, get equally cooked throughout. And the larger the mass of meat and the more closely the mass is packed into the cooking vessel, the longer will be the time necessary for the heat to penetrate into the interior of the meat, so as to ensure the complete cooking of its central parts. Hence in the case of 96 lbs. of meat, packed as the meat was packed on the "Cornwall" into a vessel of not many gallons' capacity, it may well be that pieces in the interior of the mass did not, during four hours' cooking, attain a temperature adequate to destroy the life of trichina parasites. Indeed it would surprise me to be told that such length of cooking was effectual to that end in the case of the "Cornwall" pork. For I myself can call to mind an instance in which a mass of salt beef 60 lbs. in weight, after what was believed to be thorough cooking in a closed boiler during nine hours, was found in its centre conspicuously under-done, notwithstanding that its exterior presented an appearance of over-cooking. In this connexion it may be remarked that tradition, as handed down by cookery-books, prescribes a quarter of an hour's boiling for each pound of meat to be cooked. If this estimate is to be trusted, the pork on board the "Cornwall" should have been boiled six times longer than was actually the practice; or else it should have been cooked in some fashion different from that ordinarily followed in "boiling."

Indications as to the quantity of the pork that might have been trichinised; as to the presence of the diseased portions in one or another cask; and as to possible specialty in distribution of such portions to a particular mess or messes, were sought for with much pains. It was hoped that some such indications might have been forthcoming during the above-recorded study of cooking operations, or from the dates of broachings of particular casks, and of the consumption of their contents. And, the pork having been consumed on certain Mondays while the manifestations of injury from its use were especially grouped about certain Tuesdays, there were many points of interest in the inquiry. Unfortunately, no record (as has been said) existed of the particular Mondays upon which pork had been consumed, or of that Monday whereon it was last eaten; nor was there any note of the date upon which each newly-broached cask of pork came into use. The pork (without discoverable trichinæ) in the harness cask might, it was seen, have been derived from one or another original cask, without such original cask being therefore exculpated. What became evident during the search was this: that no affirmative statement on any of the above points could be justified. Of no part of the pork, whether from one or another cask, or from one or another animal, could it be said that it was originally infective. And while a large proportion of the total supply might have been diseased without producing more extensive mischief than that actually observed, so, on the other hand, a small proportion of the supply being diseased could, if it were distributed after the usual fashion, be related to a prevalence of illness having the proportions and distribution that were witnessed during the outbreak. Efficiency of cooking could destroy, as far as it went, the dangerous quality of a *large amount* of originally infective pork; whereas the customary plan of

* Other vessels, saucepans, &c., containing dinners of other persons are, along with the pork vessel, placed for cooking on the stove. Possibly in practice the pork may occasionally make way for these vessels (or some of them), and become removed from time to time from the premier place on the stove to a situation on the hot plate remote from the central fire.

distributing the meat to the several messes might result in a *small quantity* of infective meat coming to be distributed somewhat widely. For, from an overplus service to one mess, a deficiency in service to another mess or messes is made up by bits or slices. Given a diseased joint as the portion designed for one mess, and bits of it taken as makeweights to other messes: and incidence of the resulting trichiniasis, largely upon the one mess, and to a less degree on the other messes, becomes readily intelligible.

In bringing this report to a conclusion certain matters that seem to call for attention may be briefly noted:—

Of all outcomes of this inquiry, both immediate and prospective, one of the most important is the circumstance that an outbreak of trichiniasis has in many essential features aped an outbreak of enteric fever. This occurrence is, so far as I know, without recognised precedent in this country. No doubt in other countries something similar has occurred; but in those instances, so far as we hear of them, no sooner the cause of the outbreak seems to have been hit on than abundant and undoubted symptoms of trichiniasis are found to have been observed in the patients. In the present case it was not so, for since the pathological evidence referred to was obtained I have again (along with the Medical Officer of the ship) inquired of the boys attacked, respecting the nature, order, and succession of their symptoms; and I am constrained to admit that although symptoms doubtless referable to trichiniasis could be elicited from many of the boys, still such symptoms had been seemingly very imperfectly developed. Especially is it noteworthy that in no single instance had *many* of these symptoms together been present even in a minor degree. Possibly the absence of defined symptoms of trichiniasis may have been in some degree due to the youth of the patients, for it is asserted by more than one authority that children suffer less severely from the disease than do adults. But however this may have been, it has in the end become apparent that no better designation for the majority of the cases (pathology apart) would be found than "simple continued fever." Now, heretofore, it has been the custom to regard fevers that could be thus designated as partaking of the nature of either typhus or enteric fever, and as having no particular significance of their own. But the experiences here recorded point to other states of disease as being properly described under the name of continued fever. In particular cases where the evidences of typhus, enteric, or relapsing fever are inconclusive, and in outbreaks that are not capable of being surely designated by any one of these names, trichiniasis (among other diseases) will now claim to be considered. The account that Mr. Mortimer de Brent (the Medical Officer of the ship) has given of the "Cornwall" disease, as it now presents itself to him after recognition of its parasitic nature, will be read with interest in this connexion. It will be found, along with notes on microscopic investigation of muscles of the fatal case, drawings by Dr. Cory, and temperature charts of the cases, in appendix to this report.

Another circumstance of interest is the small mortality in this outbreak. So far it is in accord with certain observations on the Continent. For although trichiniasis has on many occasions proved very fatal, nevertheless there are on record very considerable outbreaks of the disease with but trifling mortality. At Magdeburg from 1858 to 1862 300 persons suffered from trichiniasis, and two only died; at Gusten in 1861, 40 cases occurred without a single death; at Blankenburg in 1862 there were 278 cases, and only two deaths; and at Quedlinburg in 1864, of 120 patients, two only died:—a mortality, it will be observed,

in no instance so high as 2 per cent. of the attacks. Probably enteric fever in outbreaks of any magnitude rarely occurs with a mortality of less than 10 per cent. Henceforward, therefore, outbreaks of so-called enteric fever characterised by exceptional paucity of fatal cases must needs be regarded with peculiar interest.

It is very noteworthy also that no suspicion could well have arisen respecting the nature of this outbreak, if it had happened that the disease, instead of occurring on ship-board, had attacked members of several different families living in town or country. In regard of enteric or of "continued" fever prevalence, under such circumstances, it is unlikely that the meat source of the sufferers would have been inquired about at all; and had inquiry in such direction been made, it is altogether doubtful whether, in view of the small proportion of attacks to escapes, community of meat source would when found have been deemed to be evidence of material value, even if the meat had been pork. In the present instance the result of inquiry must have been negative, if it had not been for the frank co-operation of the Medical Officer of the ship, and for the fact that the community concerned was in essentials known, in every detail of its daily life, to the Captain Superintendent; and that book-records concerning most matters of primary importance were put at my disposal. In thanking Captain Morrell very cordially for the help of which every page of this report gives evidence, I feel it is proper that I should observe that, in providing the ship with the article of food that has now come into question, there was no sort of suggestion that it could be unwholesome, and that he acted, as he always does, in the interest equally of the ship and of the boys; while as regards cooking operations, he merely followed time-established precedents in most ships and in many public institutions.

Finally, with reference to the question respecting cooking that has been raised, it seems desirable that direct experiment should forthwith be made to settle what are the precautions requisite to be taken for the thorough cooking of meat in establishments wherein such food is cooked *in bulk*. And further, it deserves notice that if efficient examination of imported pork had been matter of rule and duly enforced, this outbreak would in all probability not have occurred.

APPENDIX (A).

Memorandum on Microscopical Examination, two Months after Death, of Muscles and Viscera from the Body of Richard Pierce.

Portions of the following muscles were examined microscopically.—Diaphragm, obliquus abdominis externus, psoas, intercostal (fragment), pectoralis major, biceps brachialis and heart. Specimens also of bowel contents from various portions of the intestine, and specimens from its mucous surface were submitted to investigation. The muscles generally were œdematous, much decomposed, and offensive. In colour they were no longer ruddy, but instead, livid, greenish, or even purple in hue. Notwithstanding, in most muscles (except the heart), the fibres were easily made out, and in some the transverse striæ also. The heart was the most decomposed of all the muscles, and its tissue appeared much altered. The intestines were decomposed internally, and their mucous membrane stripped readily from the subjacent tissue. Here and there the mucous membrane had disappeared. The bowel contents were extremely offensive, and in an advanced stage of decomposition.

In all the muscles examined, excepting the heart and intercostal, wandering parasites were found. They may be said to have been moderately plentiful;

one muscle only, the diaphragm, can be described as absolutely "infested" with parasites. In the intestine, no sexually mature worms could be discovered; but from the under surface of folds of its mucous membrane numerous very minute, short, hair-like bodies were obtained. Under a high power these bodies appeared to be without definite structure, with the exception perhaps of what might have been a central canal. Many were irregular in their outline, and most seemed partially decomposed. Probably they had once been brood parasites.

All the muscular parasites except one appeared dead when found, and none were encapsuled. Before dying, by far the greater number had failed to attain such size as is common in trichina spiralis at the completion of its wandering stage. In most respects, such as general appearance, shape, proportion of length to breadth, &c. they appeared identical with that parasite in its immature larval stage. As regards structure, however, these immature worms were indistinct, their interior having undergone granular change; many specimens were broken up and apparently more or less decomposed. Of the comparatively few parasites that had attained the full size of muscular trichinæ, the largest measured $\frac{1}{25}$ of an inch in length. Specimens of this size were in several instances translucent and "dropsical," being much distended by clear fluid; consequently their structure, especially that of their anterior portions, was extremely clearly defined. Herein they differed from non-dropsical specimens of similar apparent age whose structure had become indistinct owing to granular change in their interior. This dropsical condition of parasites was noticed for a short time only during the investigation. Before examination of the muscles had proceeded far, no specimens could be got that had not become opaque from granular change in their interior. Probably "dropsy" did not occur (during investigation) except in parasites that were dying or had not long been dead. In any case the number of such specimens was much restricted, and it became still further diminished by accidents due partly to thickness of cover-glasses, partly to extreme tenuity of the walls of the parasites. Under manipulation of a high power the specimens, besides being generally compressed and flattened out sometimes to the extent of nearly doubling their original breadth, became in most instances ruptured and their contents extruded. As a result of this compression and rupture of the best specimens, the structure of their anterior portions only can with any confidence be depicted.

Early in the investigation some doubt as to the identity of this wandering parasite seemed suggested in that the dropsical specimens presented, in their anterior portions, a much more defined structural appearance than that depicted by other observers in regard of trichina spiralis, and further, unlike that species as figured in books, their intestine, except indeed at its very commencement, presented little appearance of a succession of bead-like bodies, (the so-called "rosary") each with a central highly refractive spot. In this connexion too the circumstance that none of the numerous specimens observed were encapsuled, and few of them even suggestive of spiral involution, seemed to require explanation.* It was very soon seen, however, that all difficulties of the above sort might readily be explained with reference to the exceptional circumstances under which the parasites in question came to be observed. These were wholly different from conditions under which trichinæ have apparently yet been studied. Heretofore, observations of trichinæ have been pretty much restricted to those occurring in recently deceased hosts, human and other, or in pigs, the flesh of which has been artificially preserved. No information is to be had respecting the behaviour of trichinæ in muscles that have undergone, as in the present instance, during many weeks post-mortem decomposition. Brief consideration therefore of these exceptional circumstances in their bearing on the subject is necessary. Early death (on the 18th day of illness) assisted by decomposition of the host, may suffice to explain

* It will be observed that doubt as to identity of this wandering muscular parasite, if seriously raised at all, involves considerations of supreme importance. Its identity or non-identity with trichina spiralis would not alone be in question: for in view of the symptoms exhibited by Pierce and other sufferers by the "Cornwall" outbreak, possible relation of enteric fever to ravages of a muscular parasite would have to be investigated and dealt with; and current views respecting the etiology and pathology of that disease might require to be materially modified.—W. H. P.

the moderate (moderate. *i.e.*, in comparison with other observed cases) amount in the present instance of migration of parasites. It may serve also to account for the circumstance that comparatively few specimens had attained full growth as wandering parasites, that none were encapsuled, and that few exhibited any appearance of spiral involution. For a period of four to five weeks is set down as necessary for attainment by trichinæ of complete and encapsuled larval stage, and it seems that spiral involution of the worms is scarcely to be looked for short of commencement of that stage. Maturity therefore of many specimens could not be expected at the end of 18 days. Probably death alone of the host may have sufficed to stop migration, and have largely checked subsequent development of parasites, the more so if they are dependent on the general circulation for their transmission to and growth in the muscles. But what death by itself (of the host) may have been unable to effect, may well have been accomplished by death and decomposition jointly. Though death of a host may not (apparently it does not) affect the vitality of encapsuled trichinæ *that have ceased to feed*, non-nourishment of muscles and the decomposition of them which here followed death may well have caused destruction of immature parasites that had yet to undergo in their wandering further *feeding* and growth. On the other hand full-grown trichinæ, even if not yet encapsuled, to whom feeding is no longer a necessity, may (and evidently can) notwithstanding death and decomposition of their host, continue to exist in decomposing muscle long after their immature companions (which needed nourishment) have perished. In the present instance abstention from food of full-grown muscular parasites together with (instead of encapsulation) continued wandering in search (so to speak) of escape from adverse conditions, may well have sufficed after many weeks to produce in them absence from their interior of any extraneous matter, except water, and thus at length have rendered abnormally conspicuous certain details of their structure which, had they in ordinary course gone in a state of repletion into capsules, would have appeared indistinct or even altogether different. In view of all the circumstances dealt with, absence in dropsical parasites of well defined "rosary" appears of little moment; the less since more recent examination of full-grown muscular trichinæ from pigs has shown that an appearance in any great degree comparable with the "rosary" of books is by no means to be looked for. Further, the presence in these dropsical parasites of well-defined structural appearance of the anterior alimentary apparatus, instead of raising question as to identity of species, may rather be regarded as affording better opportunity than has yet been had for study of the anatomy of trichina spiralis. Whether or not the specimens mainly concerned have (as certain larvæ may be believed to do when checked at particular stages of their development *as larvæ*) skipped (or abridged) a stage (that of encapsulation) in their life history, and while continuing to exist in the decomposing muscle, have gone on toward higher development in the direction of mature sexual intestinal trichinæ, is a question that need not here be discussed. Figures illustrating points that have been referred to are submitted for use of other observers.

W. H. POWER,
ROBERT CORY.

Fig. A. A parasite from the abdominal muscle measuring $\frac{1}{12}$ of an inch in length. When found it was a good deal distended by clear fluid, but was *living*. The specimen is somewhat flattened by compression between the lens and the slide.

(*a.*) Mouth and œsophagus.
(*b*) Upper part of alimentary apparatus exhibiting indications of a rosary-like appearance, and a body with a central highly refractive spot.
(*c.*) Continuation of alimentary apparatus.
(*d.*) Represents what appeared to be a closed sac; possibly the reproductive apparatus.
(*e.*) Is seemingly a gland similar to that figured by other observers.
(*f.*) Posterior end of the worm, where was an aperture through which under pressure part of the contents of the specimen extruded, thus rendering its lower interior structure indistinct.

Fig. B. A parasite from the pectoral muscle. The specimen, which was highly "dropsical" when found, has been flattened and ruptured; its contents are extruded.

(a.) Mouth and œsophagus.
(b.) Upper part of alimentary apparatus, with bead-like body and central highly refractive spot.
(β.) Point of rupture of wall of parasite, and extruded contents.
(γ.) Anal aperture.

Figs. C^1., C^2., and C^3 represent portions of another highly "dropsical" parasite from the pectoral muscle. The specimen became during examination ruptured and its contents extruded.

C^1. Head, with mouth and œsophagus.
C^2. Middle portion of parasite (before rupture) showing indications of so-called bead-like bodies.
C^3. Posterior end of parasite after compression, which has caused extrusion at the anal orifice.

Fig. D. Anterior extremity of B. highly magnified.

(a.) Mouth and œsophagus.
(b.) Pharynx having apparently two muscles. One (c) having attachments at (1) and at (2); the other (d), external to (c), having posterior attachment at (3) and anterior attachment (4) at a point in front of the anterior attachment of (c). This muscular arrangement appears well adapted for purposes of suction.

Fig. E^1. A "non-dropsical" parasite from the diaphragm showing the relation of length to breadth as it appeared viewed under a low power.
Fig. E^2. The same as it appeared under a higher power.

APPENDIX (B).

MEMORANDUM by MR. MORTIMER DE BRENT, Medical Officer to the School Ship "CORNWALL," on the recent OUTBREAK of ILLNESS among BOYS on board that vessel.

On 23rd September 1879, seven boys complained at one and the same time of the following symptoms: pain in the belly, principally in the epigastric region, nausea, and distaste for food. Four of them complained also of vomiting, and one (Beaumont) vomited two or three times during the ten minutes he was under examination. All had brownish white-coated tongues. Several stated that already they had suffered for two or three days from looseness of the bowels; others on the contrary (including Beaumont) had been constipated. The nature of these symptoms pointed to stomach derangement, and their simultaneous occurrence in seven boys led to suspicion that contraband food of some sort, partaken of in common by all the sufferers, had been the cause of their illness. Inquiry however in this direction failed to show that they had eaten any food other than that provided on shipboard, and very soon it became apparent that these boys were attacked by illness far more serious than mere gastric disturbance.

In the course of the next few days most of the seven boys suffered from fever. Beaumont, whose case was somewhat in advance of the rest, had on the morning of 24th September, after a very restless night, a temperature (axillary) of 105 degrees; the temperatures of the others when first taken ranged from that figure down to 102. All now had diarrhœa, which in two cases was excessive. The motions began in a few cases by being loose and brown, but afterwards were found fluid and yellow. Delirium set in early in several of the cases, notably in Beaumont, who had to be tied in bed.

By this time other boys hitherto apparently unaffected, after complaining first of symptoms similar to those of the first batch of sufferers, began to develop symptoms of pyrexia; and though not a few of them suffered com-

To illustrate Mr Power's "Cornwall" Report.

Scale: the smallest divisions represent thousandths of an Inch.
the whole scale represents therefore $\frac{5}{10}$ of an Inch.

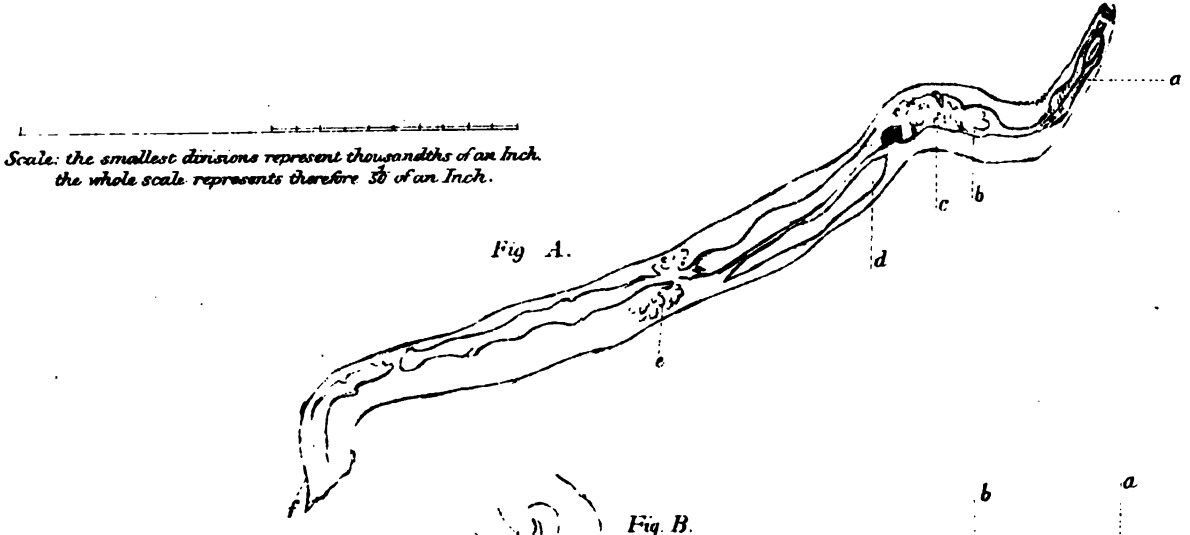


Fig A.

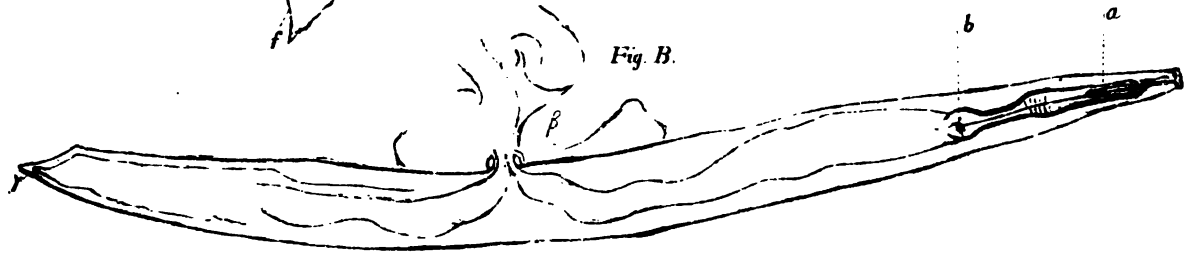


Fig. B.

Dangerfield Lith. 22 Bedford St Covent Garden

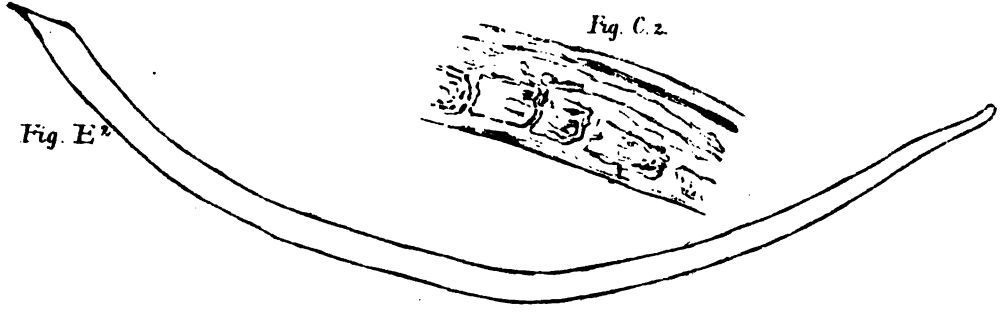
Fig. E'.

Fig. C 3.

Fig. C,

Fig. C 2.

Fig. E 2



Scale: the smallest divisions represent thousandths of an Inch,
the whole scale represents therefore $\frac{1}{50}$ of an Inch.

To illustrate Mr Bower's "Cornmill" Report.

Dangerfield Lith. 22. Bedford St Covent Garden.
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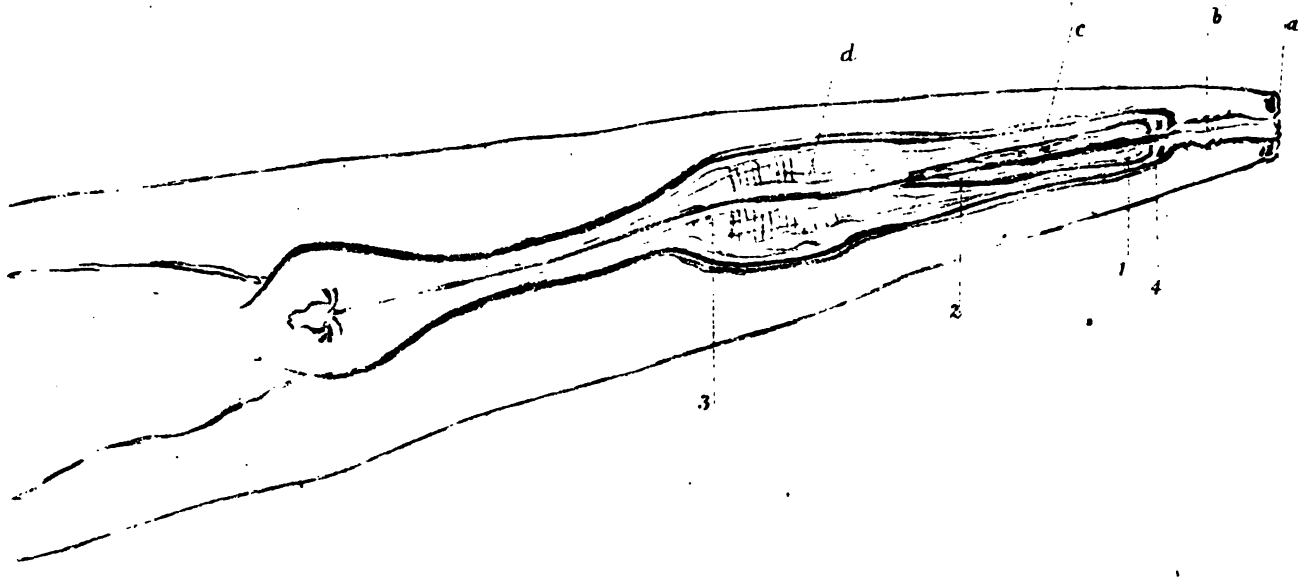
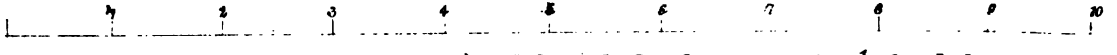
Fig D.





Scale thousandths of an Inch, the whole Scale therefore representing $\frac{1}{100}$ of an Inch.

To illustrate Mr Powers "Cornwall" Report

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paratively slightly, no doubt was felt that the outbreak was essentially one of continued fever.

In an outbreak of this sort, characterised, namely, by gastero-enteric disturbance and fever, diagnosis (and diagnosis was called for) as to its nature was based naturally enough on the symptoms of its graver cases; and three boys, Beaumont, Pierce and Sims belonging to the first group of sufferers soon afforded symptoms that seemed to point unmistakably to *enteric fever*. Besides the diarrhœa symptoms, which in Beaumont and Pierce were very severe, abdominal tenderness was observed in all cases, and in two (Beaumont and Sims) seemed to be localised somewhat in the right iliac fossa; and all three boys had tympanites. In addition there occurred in all three an eruption of roseola spots. In the case of Beaumont, in whom the eruption appeared earliest, the spots existed in considerable numbers over the abdomen on 26th September. In all respects the spots behaved like the spots of enteric fever. They could be made to disappear on pressure, and successive crops of them occurred from time to time, as was proved by marking the earlier eruptions with ink. In the case of Pierce the spots were even more abundant and extensive; Sims had about the same amount of them as Beaumont. Further, bloody evacuation occurred in two of these cases. Sims had blood in his motions once only, and then to no great amount. But Pierce had (according to the persons attending on him) copious bleeding from the bowel on three occasions toward the end of his illness; and the manner of his death was consistent with sudden hæmorrhage into his bowel as well as with (what at the time was thought to have occurred) intestinal perforation. On the evening of the 18th day of his illness he sat up in bed resting on his elbow calling for the bed-pan, and this not being brought immediately, he exclaimed "Make haste." Before however the commode could be got to the bed, "rattling in his throat" occurred, and he fell back and died.

Inasmuch as circumstances, which have recently occurred, show the diagnosis thus made to have been a mistaken one, it appears desirable, for the help of future observers, to put on record an account of the symptoms (so far as they can now be recovered) exhibited during the outbreak. And here it may be mentioned that stress of work occasioned by the outbreak, all of which along with private practice had to be borne singlehanded, has been the reason why notes of the cases have been, excepting temperatures, less complete than could be wished.

Symptoms observed during the outbreak that might have been referable to enteric fever.

The symptoms initiatory of illness, so far as the graver cases were concerned, seemed not inconsistent with commencing enteric fever. *Vomiting* is not, it is true, usually a symptom in enteric fever, but its presence in eight or ten cases was not necessarily reason for suspecting other disease. *Abdominal tenderness and pain* existed in all the severe cases, and in two seemed to be most acute in the right iliac region. *Tympanites* was observed more or less in all the cases confined to bed. *Diarrhœa:* two only of the severe cases were without looseness of bowels at some period of their illness; some had diarrhœa throughout. In two cases it was, as has been said, severe. *The motions* in the diarrhœa cases were in colour and consistency just such as might be looked for in enteric fever. *Bowel hæmorrhage:* three boys had bleeding from the bowels; in one, Pierce, the hæmorrhage was copious, and repeated on three occasions. *The tongue*, though at first coated with brownish-white fur, became during the progress of severe cases dry and brown, with reddish edges. In some cases the teeth and lips were covered with sordes. *Eruption of spots:* nine boys had spots of some sort, especially noticeable on the abdomen. In most cases they were just such spots as might have been expected in enteric fever. Certainly in one case they appeared in successive crops. *Epistaxis:* three boys had epistaxis; one, Brawnis, had it severely, but he was subject to it when well. *Bronchial catarrh*, though only noticeable in four cases, was just such as might have occurred in enteric fever. *Pneumonia:* one boy had pneumonia. *Deafness:* six of the boys who were severely ill (including Beaument, Pierce, and Sims) suffered more or less from deafness. *Delirium, &c.:* seven were delirious, two to the extent of requiring forcible restraint, and two also (Beaumont and Pierce) were for some days unconscious. *Relapse and slow*

recovery: three cases (including Beaumont) relapsed, and in seven cases recovery was very slow. *Loss of hair:* several boys during convalescence suffered from loss of hair.

Symptoms observed in one or another case of sickness that, after the nature of the disease had been ascertained, were seen to be consonant with symptoms of trichiniasis, as described in books.

Initiatory symptoms.—Pain in the stomach, nausea, and distaste of food were complained of in all cases from the severest to the slightest. Such symptoms are certainly in accord with those described as belonging to the commencement of trichiniasis, and their presence in *all* cases (including the doubtful ones which except for these subjective symptoms did not appear ill at all) may now be regarded as fitting better with trichiniasis than with enteric fever. The presence too of *vomiting* in the severe cases is suggestive rather of the former than of the latter affection. *Abdominal pain, tenderness, and fulness,* which existed in all the severe cases, and in many of the slighter ones also, and which commonly was a very prominent and troublesome symptom, is in equal accord with a theory of trichiniasis and of enteric fever. So also is the *tympanites. The diarrhœa* again, which was a conspicuous feature of the severe cases, and which was doubtfully present or even absent in the slighter ones, might as well have belonged to trichiniasis as enteric fever. The character of the *evacuations* too is compatible with the affection having been trichiniasis. Blood in the stools has not however heretofore (so far as I know) been noted in trichiniasis. The *tongue* furred and brownish-white at the commencement (and throughout in slight cases), and becoming dry and brown with reddish edges during the progress of severe cases, was related probably rather to alimentary disturbance and fever than to specific disease of either sort. The same remark applies to *sordes. The temperatures* were none of them in any way typical of enteric fever; and this is especially to be remarked in the inception of the curves; moreover, in many and in severe cases the difference ordinarily to be observed in enteric fever between the morning and evening temperature was not noticeable. In short the temperatures merely showed "continued" and in severe cases long-sustained fever. *Sweating,* which is mentioned as occurring in trichiniasis, was present in several severe cases. In Beaumont and Bradshaw it was profuse and wetted the bedclothes. *Eruption of roseola spots.* Excepting boils, eruption of any sort in trichiniasis appears to be unknown. As regards the outbreak in question it deserves notice that in more than one case having enteric-like eruption, the spots were unusually large and blotchy; and that in one instance (Pierce) if not in others, the spots abounded on the arms, thighs, and legs as well as on the chest and abdomen. *Bronchial catarrh, pneumonia* (one case), *deafness,* and *delirium* in certain of the severe cases, are symptoms compatible with the outbreak having been trichiniasis. So also was the occurrence of *relapses* and the *loss of hair* during convalescence. The *duration of illness* may now seem to favour the view of trichiniasis. It differed greatly, varying from ailment for a few days in slight or doubtful cases to illness lasting without break (except temporary temperature fall) for upwards of 70 days in the case of Sims. *Jaundice* occurred during and after the outbreak in some dozen boys, few of whom had even trifling indisposition. Though not known heretofore to be thus related to outbreaks of either enteric fever or trichiniasis, its occurrence under present circumstances deserves notice, as having possibly parasitic cause.

Symptoms that might in an outbreak of trichiniasis be expected to manifest themselves, but which in the "Cornwall" outbreaks were by no means prominent.

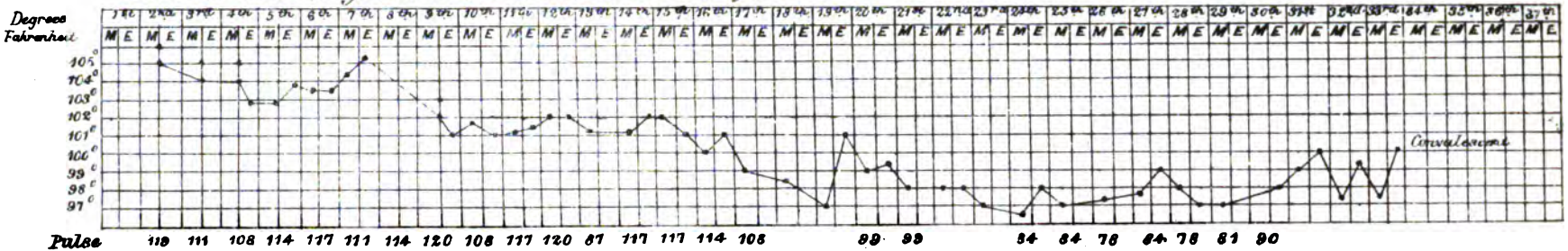
Pain in the limbs early in illness was not complained of, except perhaps in one case (Brace), who is entered in the day-book as having "rheumatism." Another boy is remembered on falling ill to have moved as if suffering from stiffness. *Œdema of the face or eyelids* was noticed in only two instances. Many of the boys, indeed, had flushed faces on falling ill, and about the eyes looked as if suffering from bad cold in the head. *Muscular symptoms.*—In no case was obvious *swelling* of muscles present. *Contraction* of muscles might have existed in one case (Wear), who lay in bed with his knees drawn up to his chin, and with his fore-arms flexed. He was this time

APPENDIX C.

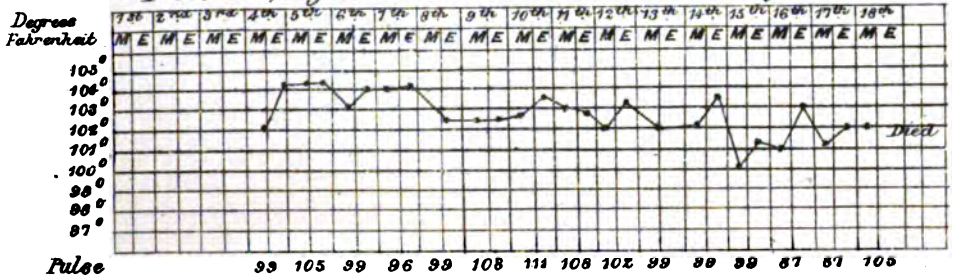
CHARTS OF THE TEMPERATURES RECORDED IN 20 CASES OF ILLNESS OCCURRING ON BOARD THE "CORNWALL."

(*The tops of the columns are numbered consecutively from left to right with reference to the days of treatment; not in regard of days of the month. M represents morning and E evening temperature. The figures to the left of the columns represent degrees Fahrenheit; those at the bottom of the columns, pulse beats per minute.*)

Beaumont, Aged 15. Under treatment 42 days

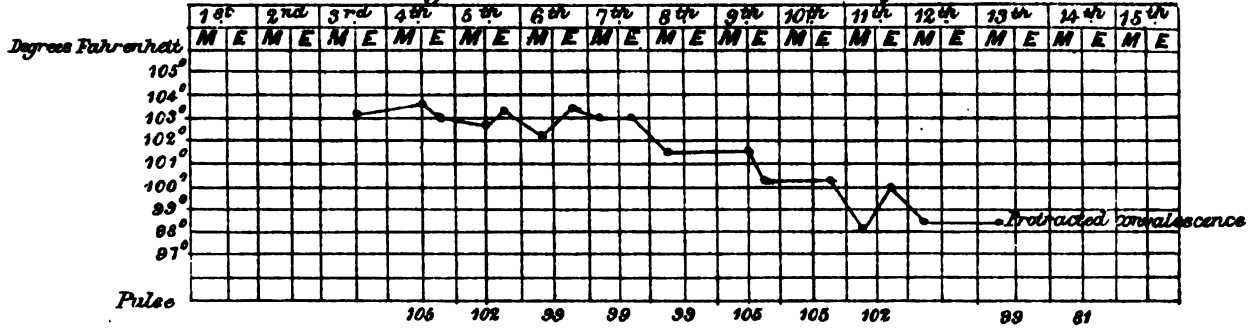


Pierce, Aged 16. Under treatment 18 days (Fatal case.)

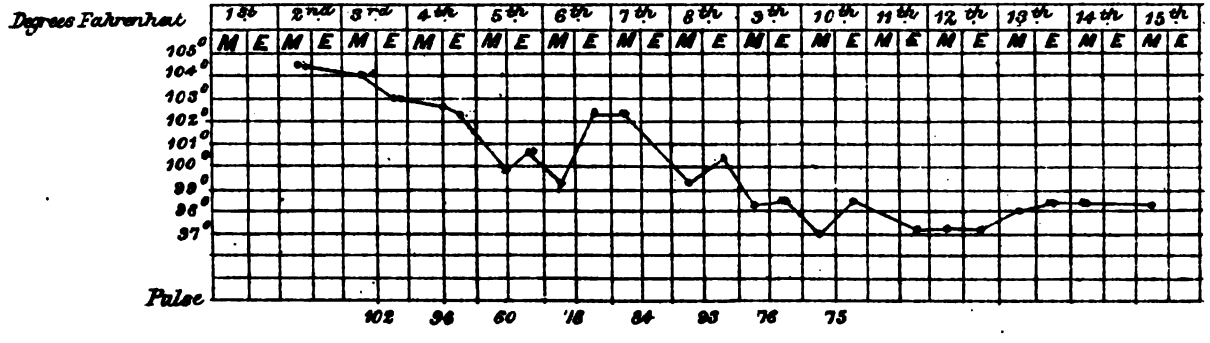


DANGERFIELD, LITH. 22 BEDFORD ST COVENT GARDEN.

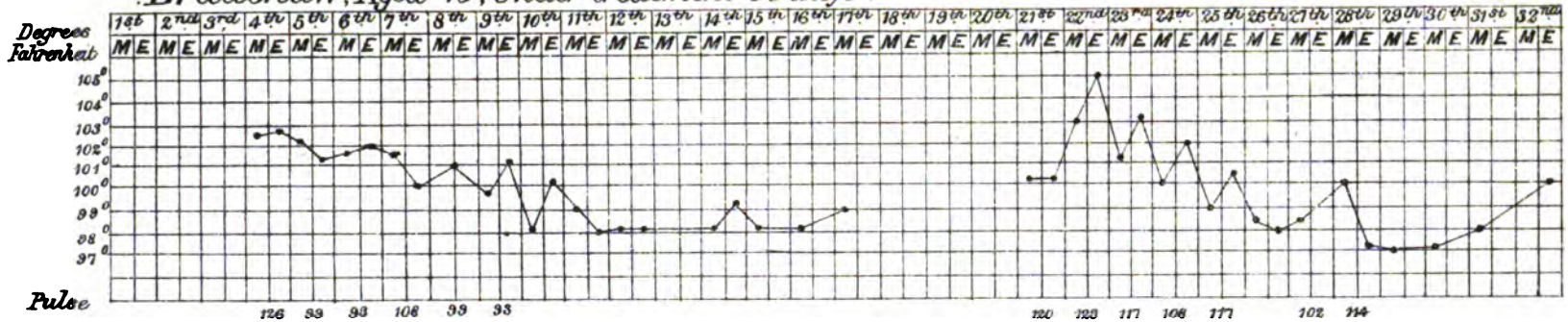
Wheeler, Aged 13. Under treatment 37 days.


1st 2nd 3rd 4th 5th 6th 7th 8th 9th 10th 11th 12th 13th 14th 15th
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
Degrees Fahrenheit
105° 104° 103° 102° 101° 100° 99° 98° 97°
Protracted convalescence
Pulse
105 102 99 99 99 105 105 102 99 81

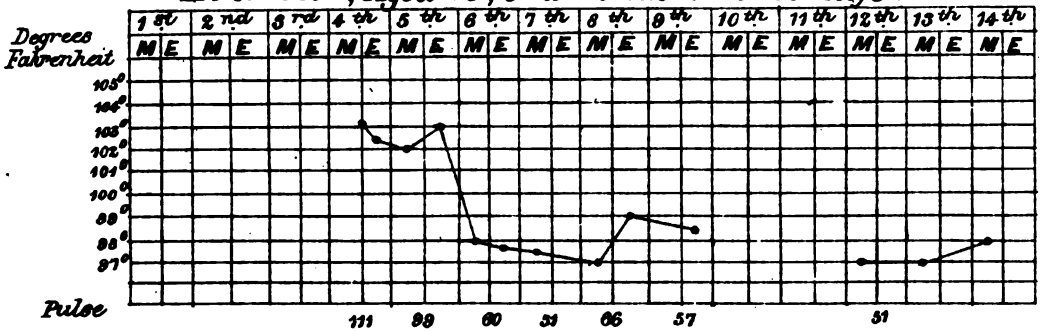
Davis, Aged 16. Under treatment 28 days.


1st 2nd 3rd 4th 5th 6th 7th 8th 9th 10th 11th 12th 13th 14th 15th
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
Degrees Fahrenheit
105° 104° 103° 102° 101° 100° 99° 98° 97°
Pulse
102 96 60 78 84 93 76 75

Bradshaw, Aged 16, Under treatment 38 days.



Brawnus, Aged 16, Under treatment 26 days.



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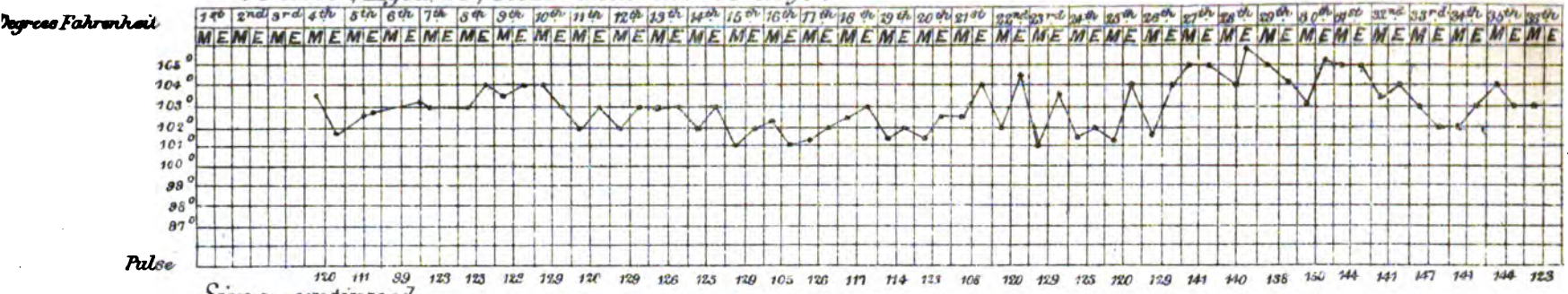
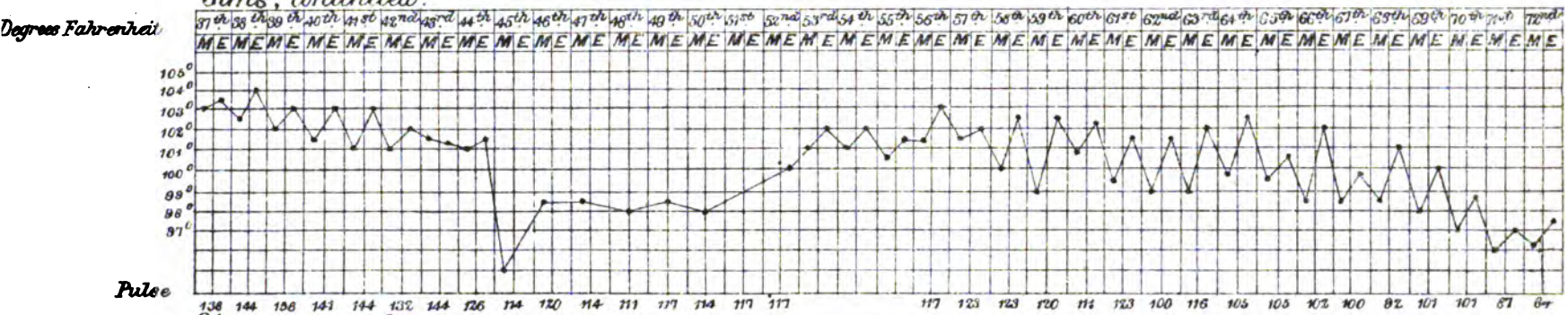
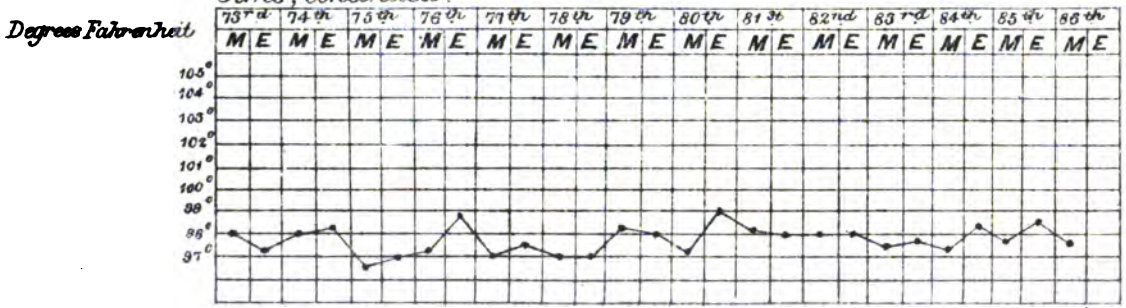
Sims, Aged 15, Under treatment 88 days.



Sims, continued.

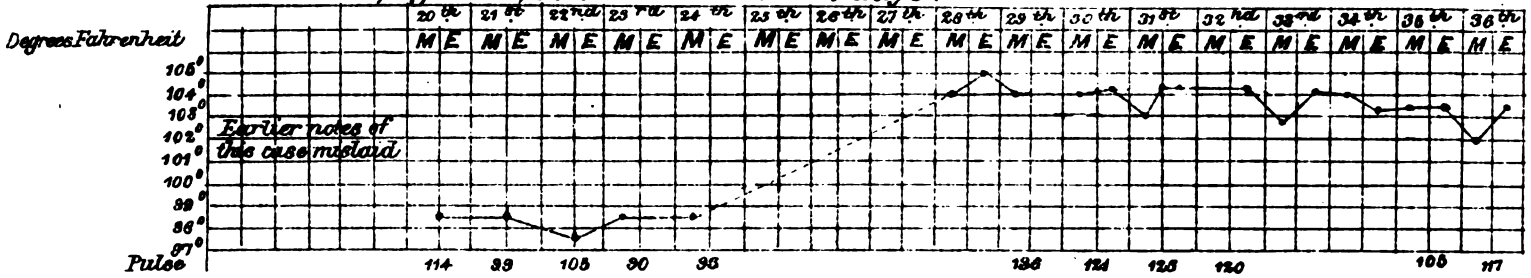


Sims, continued.

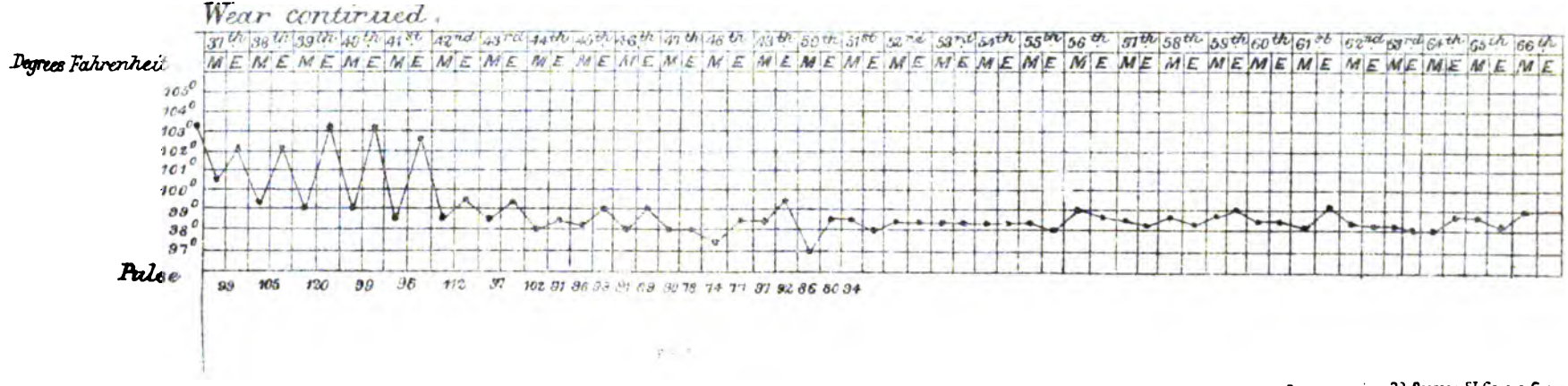


Dangerfield, Lith. 22 Bedford St Covent Garden

Wear, Aged 15, Under treatment 67 days.


Degrees Fahrenheit
105°
104°
103°
102°
101°
100°
99°
98°
97°
Earlier notes of this case mislaid
20th 21st 22nd 23rd 24th 25th 26th 27th 28th 29th 30th 31st 32nd 33rd 34th 35th 36th
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
Pulse
114 99 105 90 96 136 124 128 120 105 117

Wear continued.


Degrees Fahrenheit
105°
104°
103°
102°
101°
100°
99°
98°
97°
37th 38th 39th 40th 41st 42nd 43rd 44th 45th 46th 47th 48th 49th 50th 51st 52nd 53rd 54th 55th 56th 57th 58th 59th 60th 61st 62nd 63rd 64th 65th 66th
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
Pulse
99 105 120 99 96 112 97 102 91 86 93 91 69 80 78 74 77 97 92 86 80 94

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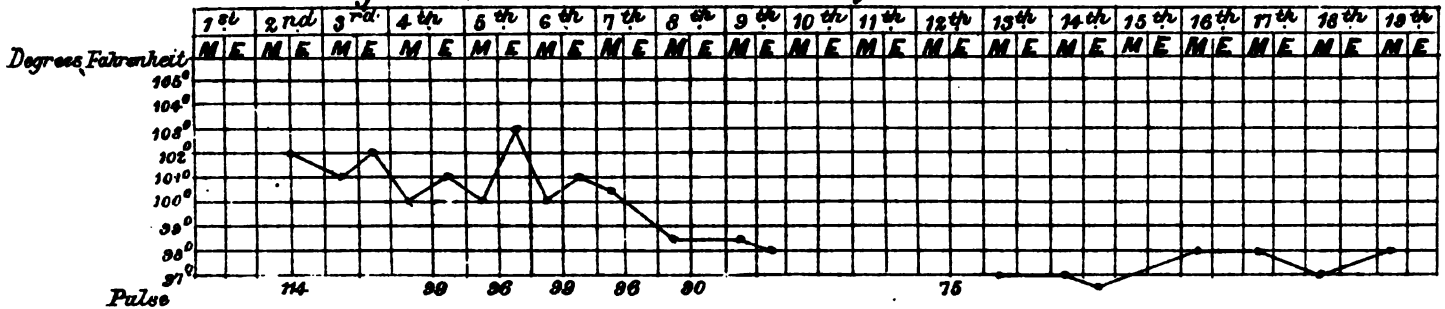
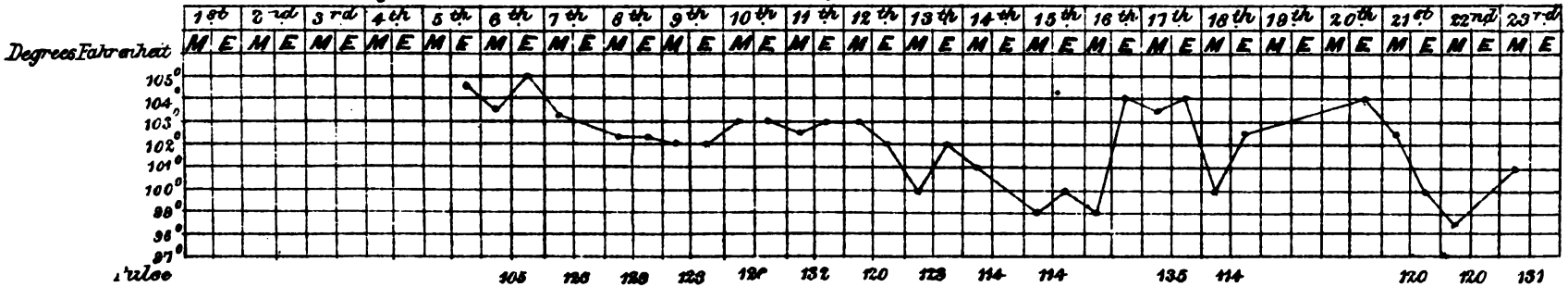
Putt, Aged 15, Under treatment 29 days.



Howes, Aged 14, Under treatment 35 days.



Dangerfield, Lith. 22, Bedford St Covent Garde
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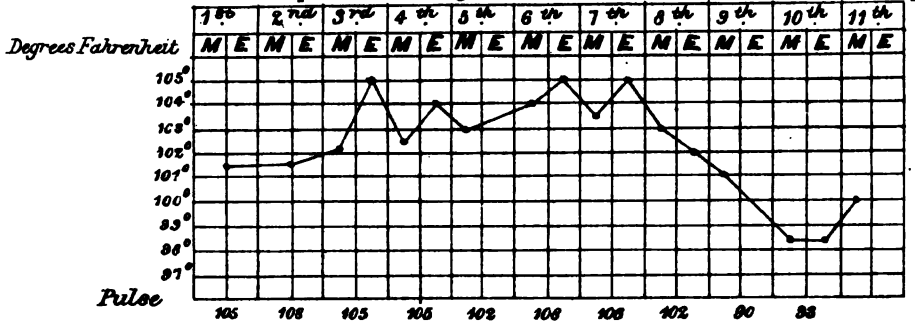
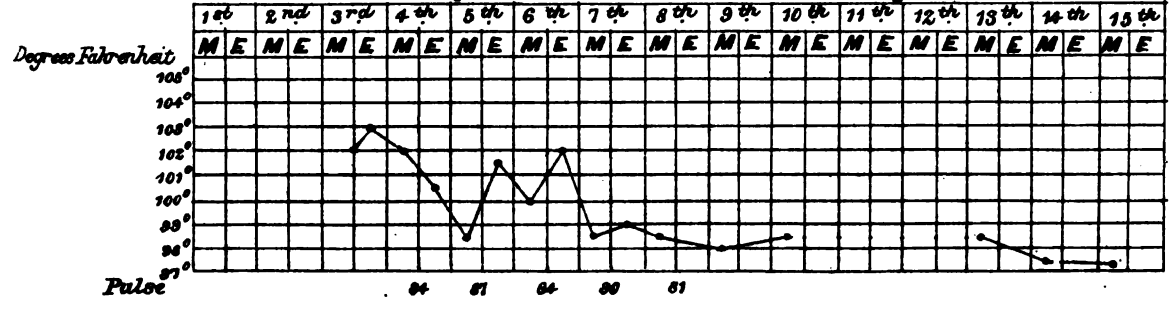
Hopcroft, Aged 15, Under treatment 18 days.


1st 2nd 3rd 4th 5th 6th 7th 8th 9th 10th 11th
M E M E M E M E M E M E M E M E M E M E M E
Degrees Fahrenheit
105° 104° 103° 102° 101° 100° 99° 98° 97°
Pulse
105 108 105 105 102 108 108 102 90 98

Salmon, Aged 16, Under treatment 18 days.


1st 2nd 3rd 4th 5th 6th 7th 8th 9th 10th 11th 12th 13th 14th 15th
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
Degrees Fahrenheit
105° 104° 103° 102° 101° 100° 99° 98° 97°
Pulse
84 81 84 89 81

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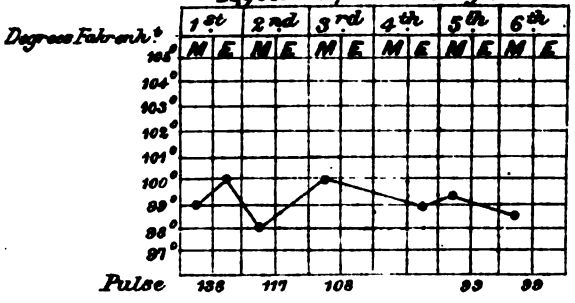
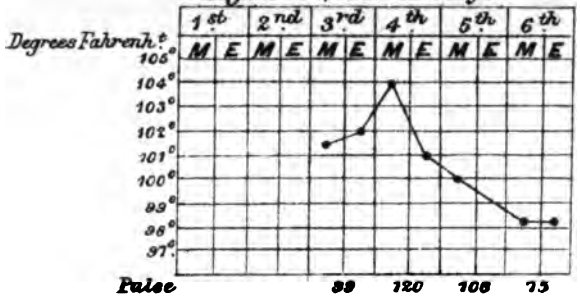
Rogers.

Aged 14, ill 8 days.



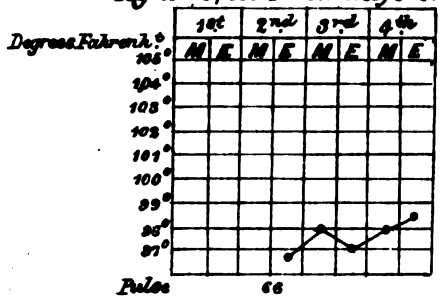
Speer.

Aged 17, ill 18 days.

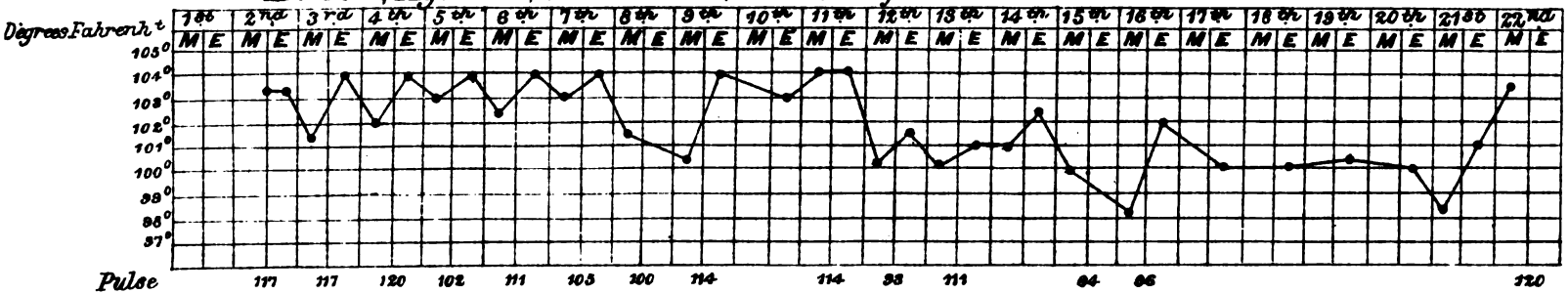


O'Hara.

Aged 16, ill a few days only



Brice, Aged 16, under treatment 31 days.



Dangerfield. Lith. 22. Bedford St Covent Garden.
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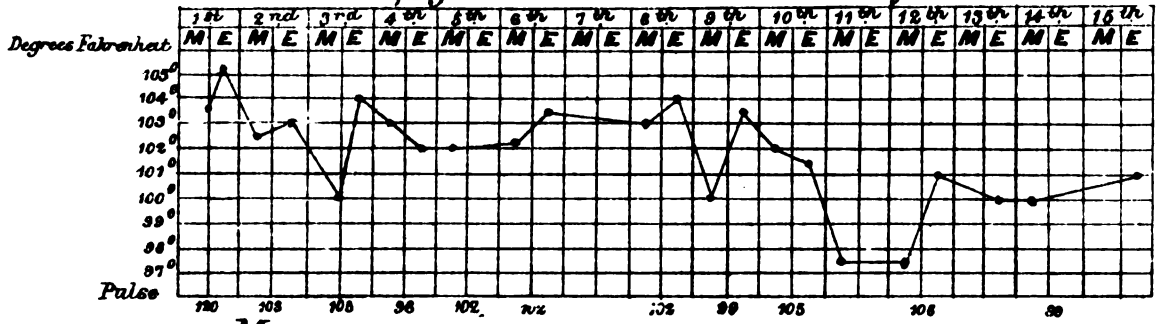
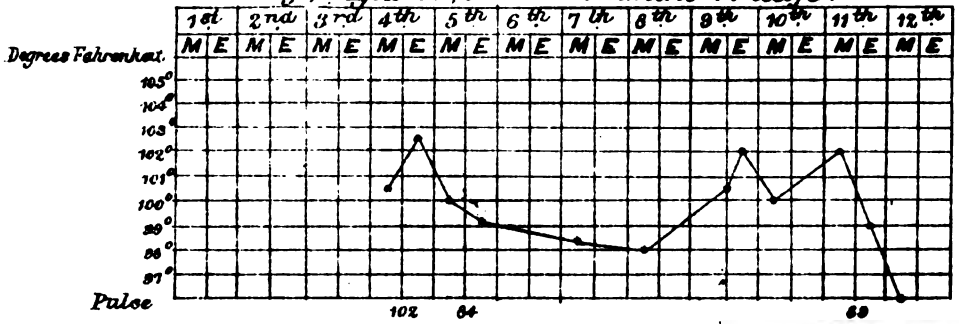
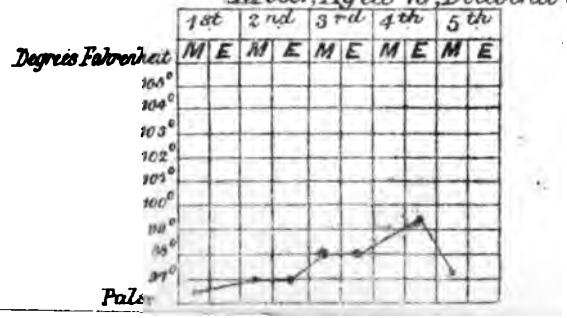
Bowler, Aged 15. Under treatment 18 days.



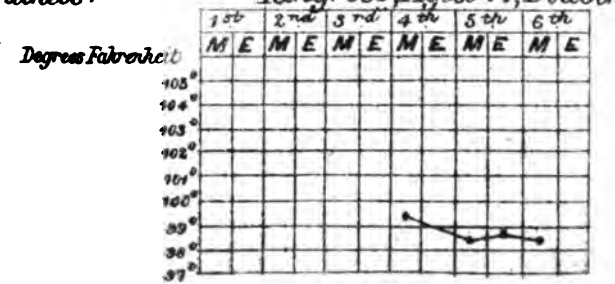
May, Aged 15, Under treatment 14 days.



Muir, Aged 15, Doubtful illness.



Ringrose, Aged 14, Doubtful illness.



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only semi-conscious, and made no complaint whatever. Several others of the boys on moving about during early convalescence appeared to suffer from stiffness; but it is difficult to estimate how far this may not have been due to confinement to bed. *Tenderness and soreness of muscles* was not conspicuous. Possibly the abdominal tenderness, so noticeable in many cases may have been partly due to soreness of the muscles. In one case (Marks, who had pneumonia), the muscles of the chest were decidedly tender to touch and afterwards this boy suffered from soreness of the left biceps. Several boys, too, as the attendants of the sick now remember, cried out with pain on being moved in bed. In Sims this was specially noticeable, voluntary movement of the head, in his case, on the pillow, causing exclamations of pain. *Distressing dyspnœa, trismus, marked aphonia,* or *impaired movement of the tongue,* were not noticed in any case. *Dysphagia,* however, may have been present in Pierce. He complained constantly of sore throat, and as no objective symptoms could be discovered therein, this may have been due to tenderness or soreness of the muscles of deglutition. *Œdema of the feet, legs, and thighs* was not present except in one case (Marks). This boy had an attack of pneumonia, during which he suffered early and continually from epigastric pain, complained also of nausea, and could not be got to take his food. He it was who had tenderness of the thoracic muscles, and afterwards soreness of the biceps. *Anasarca of the trunk* was absent in all cases.

No. 7.

App. A. No. 7. On some Nematoids found in the Body of a Boy who died from an Epidemic Disease (supposed to be Trichiniasis) on board the Reformatory School Ship "Cornwall," by Dr. H. Charlton Bastian, F.R.S.

On Nematoids from *Cornwall* "fever" case, by Dr. Bastian.

This report was undertaken at the request of the Medical Officer of the Local Government Board. I was asked to examine the drawings and any specimens still existing of the Nematoids found in the body of the boy who died in the autumn of 1879 on board the "Cornwall," with a view to define the species or relations of this parasite. I was also requested to state "what has heretofore been known respecting its habitat "and habits in one and another stage of its existence."

In illustration of the nature of the epidemic disease supposed to have been occasioned by these Nematoids, it will be well for me to recapitulate the following facts and opinions from the careful report of Mr. Power—especially as they have some bearing upon the question as to the precise nature of the organisms themselves.

The occupants of the "Cornwall" at the time of the epidemic were 262 boys and 15 officers—the age of the boys ranging from 12 to 20 years. Between the 23rd September and the 23rd October 1879, 43 of these boys were attacked with a febrile illness of varying degrees of severity. Of the boys who were attacked, only one died.

In reference to the characters of the disease the following quotations may be made from Mr. Power's report. He says: "There can be "no doubt that all the serious cases, and some of the slighter ones "as well, had a disease which was properly described as a 'continued "fever.' In most instances the symptoms were clearly indicative of "enteric disturbance, and in some cases there were in addition, per-"sistent diarrhœa, rose-coloured spots on the abdomen, and hæmorrhage "to a greater or less extent from the bowels. From these data, the "outbreak was considered one of enteric fever, presenting perhaps an "unusual number of abortive cases, and as possibly complicated by "malingering."

Subsequent inquiry tended to discredit the notion that the epidemic was of the nature of typhoid fever, and led to the suspicion that it might have been one of trichiniasis. No distinct evidence could be produced that trichinised food had been taken, though it seemed possible that a particular pork-supply was related to the distribution of the disease. A careful examination, moreover, of all the clinical records that could be obtained of the illness, though, as Mr. Power says, "It failed to elicit "information directly suggestive of trichiniasis, failed to a like degree to "support a theory of enteric fever. . . . . If the outbreak had been "one of trichiniasis, certain symptoms usually described as characteristic "of that disease, had not been prominent. Notably, 'sudden swelling "of the face, particularly of the eyelids, at an early stage of illness,' "had not, except perhaps in one or two instances, been noticed. No "obvious 'painfulness and immobility of the arms and legs, with "œdema and contraction of the muscles' could, except doubtfully in a "single instance, be heard of. Again, 'œdema of the feet, legs, and "thighs towards a later stage of illness' had not, except in one case, "existed. General 'anasarca of the trunk' had been altogether "absent. But, nevertheless, it became apparent that though in no "single instance had all the symptoms referable to trichiniasis been "present, still in several cases one or more symptoms that might

"have belonged to that disease had existed, and in other cases such "symptoms seemed obscurely hinted at. Further in some half dozen "instances there had been jaundice of an anomalous sort. Upon the "whole it did not seem that trichiniasis could, from this clinical "history, be set aside; though evidence affirmative of it was thus far "wanting. On the other hand, these new details left me unconvinced "that the outbreak had been enteric fever; and this, notwithstanding the "occurrence in certain cases of symptoms more or less diagnostic of "that disease. . . . . It became indeed increasingly evident that "a large proportion of the fever cases might be more safely ranged "under some general name, such as 'simple continued fever,' than "under the specific name of enteric fever; and it was observed, too, of "the less febrile cases, that they, like the more febrile, were accom-"panied by pain in the belly, and nausea, with, though commonly "without, looseness of the bowels."

Under these circumstances it was at last determined to seek for some more direct evidence as to the nature of the disease, by exhumation and examination of the body of the only boy who had died. Mr. Power says:—"This course had the additional advantage of dealing with what "was in effect a test case of the outbreak. The case in question was "that of the boy Pierce, who was one of the first group of sufferers "attacked on September 23rd. He in particular exhibited symptoms "(persistent diarrhœa, copious eruption of rose spots, and even free "hæmorrhage from the bowels) believed at that time to be those of "enteric fever. In addition, he, in dying on the 18th day of his illness, "appeared to be the subject of bowel perforation."

The body of this boy was accordingly exhumed and examined by Mr. Power and Dr. Cory on December 6th, that is, just two months after its burial. Mr. Power says:—"The various viscera were easily "made out, and so far as naked eye appearances went, were not much "changed. Beyond enlargement of the spleen, which was double the "normal size, there was nothing suggestive of enteric fever. There "was no peritonitis nor any evidence of perforation. The peritoneum "generally was clear, shining, and free from lymph or adhesions. No "enlargement of the mesenteric glands could be discovered. The "stomach, intestines, some of the other viscera, and portions of various "muscles were reserved for further and more exact examination."

This further exact examination revealed, Mr. Power says, "no such "anatomical changes as are characteristic of enteric fever towards the "end of the third week of its duration," so that, "the conclusion now "seemed warranted that, whatever had been the nature of this boy's "fatal illness, it certainly had not been enteric fever."

Mr. Power adds:—"This conclusion was more than confirmed by the "results of microscopic investigation conducted with reference to "trichiniasis. In the very first specimen examined, a few fibres from "one of the abdominal muscles, was found a *wandering and living* "*trichina;* and further search revealed the presence of these parasites "in most of the muscles examined. Although tolerably abundant, the "trichinæ could not be said to 'infest' (in the common sense of the "word) any of the muscles, except perhaps the diaphragm, and in none "of them had the parasite reached the stage of encapsulation. Except "the first found, no trichinæ exhibited active movement, and com-"paratively few had attained, as had that one, their full growth as "muscular parasites. In specimens of this sort the internal structure "and organization were under a high power ($\frac{1}{1}\frac{1}{2}$) readily made out, the "parasites having altogether resisted decomposition, and most of them "having probably died only a short time before examination. The

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"smaller, not fully grown, muscular trichinæ, on the other hand, had "seemingly all long since died. Their outline had become indistinct, "their interior granular, and their structure could not be made out. "Many were found broken up. Examination of the decomposing "contents of the small intestine failed to find any parent trichinæ, but "by scraping the under surface of the folds of mucous membrane of the "bowel, brood trichinæ* in considerable numbers were obtained. These "had apparently all died, and their structure could not satisfactorily be "made out."

"Doubt could no longer exist that this boy died from acute trichiniasis. "Not only was the presence in his body of the actual parasite demon- "strated, but the results of microscopical examination pointed to the "parasites as having played a very active part in the production of his "illness, and were indeed precisely such as might have been anticipated "in a case of early death from trichina disease. Death took place in "this instance on the 18th day after appearance of symptoms, at a "period that is too short to have allowed of universal migration of brood "trichinæ, and insufficient also for the attainment of full growth in the "muscles of any but early migrants. At the same time the period was "not nearly long enough to have sufficed for the full evolution and "encapsulation of many (if, indeed, of any) muscular parasites."

In Appendix A. to Mr. Power's report further details are given by himself and Dr. Cory concerning the sites and characters of the parasites found in the body of the boy Pierce. This Appendix is, moreover, illustrated by certain figures. From it, the subjoined quotations are taken:—

"Portions of the following muscles were examined microscopically: Diaphragm, obliquis abdominis externus, psoas, intercostal (fragment), pectoralis major, biceps brachialis and heart. Specimens also of bowel contents from various portions of the intestine, and specimens from its mucous surface were submitted to investigation. The muscles generally were œdematous, much decomposed, and offensive. In colour they were no longer ruddy, but instead, livid, greenish, or even purple in hue. . . . . In all the muscles examined, excepting the heart and intercostal, wandering parasites were found. They may be said to have been moderately plentiful; one muscle only, the diaphragm, can be described as absolutely 'infested' with parasites. In the intestine, no sexually mature worms could be discovered; but from the under surface of folds of its mucous membrane numerous very minute short hair-like bodies were obtained. Under a high power these bodies appeared to be without definite structure, with the exception, perhaps, of what might have been a central canal. Many were irregular in their outline, and most seemed partially decomposed. Probably they had once been brood parasites. . . . . All the muscular parasites except one appeared dead when found, and none were encapsuled. Before dying, by far the greater number had failed to obtain such size as is common in trichina spiralis at the completion of its wandering stage. . . . . Of the comparatively few parasites that had attained the full size of muscular trichinæ, the largest measured $\frac{1}{22}$ of an inch in length."

In the microscope slides handed over to me for examination by Dr. Cory, I found four nematoids, and of them one only, a nearly mature male, was uninjured. One of the damaged specimens seemed to

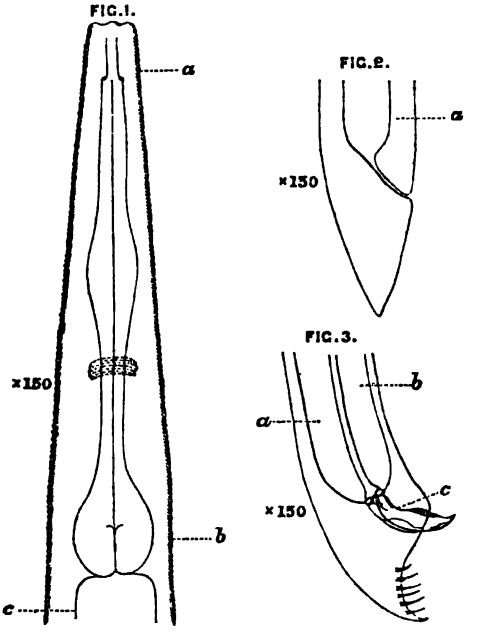
* The nature of these bodies is spoken of somewhat more doubtfully in the Appendix, as will be seen from a passage quoted below.—H.C.B.

be a not quite mature female, and from this specimen certain anatomical characters were still to be made out. The other two, partly from imperfect preservation and partly from extent of actual damage, were useless for the purposes of the present inquiry.

An examination of the two specimens above-mentioned, as well as of the drawings given by the previous reporters in the Appendix, have convinced me that the parasite is not trichina spiralis, but a new nematoid not previously encountered within the human body.

Muscle-trichinæ are sexually immature forms, about the same size, it is true, as the larger of the nematoids found in the "Cornwall" boy. These latter, however, are more or less sexually mature forms, measuring about $\frac{1}{2}\frac{1}{1}$ of an inch in length, by $\frac{1}{500}$ in breadth; whereas, of the sexually mature trichinæ, the length of the female is said to be $\frac{1}{8}$ of an inch, and the length of the male $\frac{1}{18}$ of an inch.

Again, the posterior extremity of the male trichina is bilobed, and furnished with a single terminal V-like spicule.* That of the new nematoid is altogether different, and is furnished with two sub-terminal and rather large spicules (fig. 3). The posterior extremity of the female trichina is bluntly rounded, the anus is terminal, and the vulva is



Pelodera setigera, n. sp.

Fig. 1.—Anterior extremity of female; showing tubular pharynx and two œsophageal swellings, the tube between them being crossed by a very delicate ring of nerve (?) tissue [made much too dark in wood-cut]. *a*, level at which lateral pores are situated; *b*, level of ventral pore; *c*, commencement of intestine.

Fig. 2.—Posterior extremity of female. *a*, terminal portion of intestine.

Fig. 3.—Posterior extremity of male. *a*, intestine; *b*, genital tube; *c*, spicules. (Very delicate striation of integument indicated only in Fig. 1.)

situated at the end of the anterior $\frac{1}{5}$ of the body; in the new nematoid the posterior extremity (fig. 2) is pointed, the anus is sub-terminal, and

* See for figures Leuckart, "Die Menslich. Parasit," Bd. ii., figs. 291–293; and Schneider, "Monograph der Nematod," tab. xiii., fig. 1.

the vulva is situated near the centre of the body. In addition to these differences, the shape of the œsophagus is altogether dissimilar in the two forms, and the rosary-like arrangement of the anterior half of the alimentary canal (produced by an investment of peculiarly large cells similar to those also met with in *Trichocephalus and Trichosoma*) is altogether absent in the new organism.

The new nematoids, in fact, seem, as far as I can ascertain, to present all the characters which distinguish the free nematoids (Anguillulidæ) from their more habitually parasitic allies. Elsewhere* I have pointed out the nature of these distinctive characters, and have shown that they are furnished by the conformation and arrangement of the male and female genital organs,† "the males having two equal sub-"terminal intromittent spicules, with or without accessory pieces, "whilst the females have the vulva situated at about the middle of the "body, a short vagina with a symmetrical double uterus, whose branches "lie on opposite sides and are connected with a short and simple "reflexed ovarian tube—the only exception to this arrangement being "in a few genera, in which the females having the vulva situated some "way behind the middle of the body (about the commencement of the "posterior third), the hinder segment of the uterus remains abortive "and undeveloped."‡

Of the free nematoids already known, the new form is most closely related to those of the genus *Rhabditis* as newly defined by myself, and to those of the genus *Pelodera* (Schneider). Its affinities with the latter genus are, however, by far the most numerous.

The representatives of both these genera possess the same shaped pharynx and œsophagus as we get in the new nematoid. But its lipped anterior extremity, the almost certain existence of a central inferior pore nearly opposite the posterior extremity of the œsophagus, the shape of the male spicules, together with the absence or indistinct nature of the accessory piece, and the mode of grouping of the setæ on each side of the posterior extremity of the male, are all of them characters by which the new form shows its affinities to *Pelodera* rather than to *Rhabditis*.§ In the single male of the new form no membranous alæ can be recognised; but it seems possible that, though they are now no longer distinguishable, they may have existed during life and when the animal was in a better state of preservation.

Should the caudal alæ prove hereafter to be really wanting, so that setæ exist alone without lateral membranes, as shown in fig. 3, it would seem probable that for this and other reasons the new nematoid would

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* Monograph on the Anguillulidæ, Trans. of Linn. Soc., vol. xxv., p. 76, 1864.

† For figures see Philos. Trans., MDCCCLXVI., pl. xxviij., figs. 1 and 2.

‡ This particular combination of characters is only *possibly* possessed by two of the genera of parasitic nematoids described by Dujardin in 1845 in his *Histoire naturelle des helminthes*, viz., in Leptodera and Angiostoma. These two types, though apparently very different from one another, have been included by Schneider (*Monographie der Nematoden*, 1866, pp. 154-162), together with certain free nematoids, in the single genus *Leptodera*. Though dissimilar forms are, as it seems to me, thus brought too close together, the proceeding shows that the relations of the forms included by Dujardin in his genera *Leptodera* and *Angiostoma*, with some of those in his genus *Rhabditis*, was fully and independently recognised by Schneider, as it had been by the present writer.

§ For figures of *Pelodera* see Schneider's *Monograph der Nemat.*, tab. x., fig. 9, and tab. xviij., fig. 2; and for those of *Rhabditis* see writer's Memoir in *Trans. of Linn. Soc.*, 1864, tab. x., figs. 60-70. Neither lateral vessels nor an œsophageal nerve ring have been recognised in *Rhabditis*, though they are both figured by Schneider in *Pelodera*, and I have likewise found indications of their existence in the new nematoid.

have to be ranged in a genus apart, but for the present it should certainly be regarded as a member of the genus *Pelodera*. It may be appropriately enough named *Pelodera setigera*, and its characters so far as they can at present be made out, may be thus defined.

PELODERA SETIGERA, n. sp.

Female (probably immature).—Length $\frac{1}{25}''$, breadth $\frac{1}{500}''$.

External Characters.—Body white, tapering gradually towards anterior extremity, but very abruptly at posterior extremity, which is pointed. Head not truncated, but lipped. Integument with very delicate transverse striæ, about $\frac{1}{1000}''$ apart.

Pharyngeal Cavity cylindrical, $\frac{1}{1000}''$ long by $\frac{1}{6000}''$ broad. *Œsophagus* about $\frac{1}{5}$th of total length, provided with a median and a terminal swelling, and encircled just beyond the former by a delicate band of nervous (?) tissue. *Intestine* covered apparently with cells, containing only colourless particles. *Anus* $\frac{1}{400}''$ (?) from posterior extremity. *Vulva* slightly posterior to middle of body. *Ventral pore* (?) opening near posterior extremity of œsophagus. Circular *lateral pore* on each side, (as in genus *Plectus*) nearly opposite termination of pharynx.

Male (mature or nearly so).—$\frac{1}{33}''$ long by $\frac{1}{5000}''$ broad. *Spicules* of faintly brownish tint, $\frac{1}{500}''$ long by about $\frac{1}{2700}''$ in maximum breadth; no accessory piece recognisable. *Alæ* not distinct, possibly absent. About six stout *setæ* on each side of the posterior extremity of the body, not reaching forward as far as the spicules, the larger of the setæ being $\frac{1}{3000}''$ in length.*

Habitat.—In the muscles of a human corpse which had been buried for two months.

In regard to the ordinary distribution and habitats of the *Anguillulidæ* I have elsewhere said:†—"I am inclined to believe that these free "nematodes will be found to constitute one of the most widely "diffused and numerically abundant groups in the whole animal king"dom, rivalling, in the first respect at least, the ubiquitous diatoma"ceæ." Thus the land and freshwater species were found "in all he "specimens of soil examined; in moss, various species of lichen, about "the roots of fungi, also the roots of grasses and between the sheaths "of their leaves, amongst the mud of ponds and rivers, on the fresh"water algæ, amidst decaying liverworts and mosses, and on submerged "aquatic plants. The marine species exist in great abunpance in the "surface mud of rivers and estuaries, in the sand, and amongst the small "stony débris under the shelter of rocks as well as in the tide-pools, "where they swarm about the roots of the corallines, and on some of "the smaller and finer sea-weeds, especially those having dingy ap"pearance from the presence of diatomaceæ. And, lastly, two or three "species I have found in the greatest abundance, as pseudo-parasites, "within the substance of some of the softer sponges."

The marine and fresh water forms are usually distinct from one

* This distribution of the setæ (as seen laterally) seems almost similar to the arrangement in *Pelodera*, as depicted from the ventral aspect by Schneider (*loc. cit.* tab. 10, fig. 9*c*.). The two small setæ represented by him as situated further forward in *P. strongyloides* are, if present, not recognisable in *P. setigera*. In *Rhabditis*, however, the nine rays on each side are grouped in sets of one, two, and two threes—that is, in quite a different manner.

† Loc. cit. p. 25.

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another. Still, in this respect the genus *Rhabditis* is exceptional, the representatives of some of its species being found in land and fresh water, and of others in marine habitats.

In the genus *Pelodera*, Schneider included three forms; all of them are recorded as being found by him "in damp earth and decaying substances." He discovered that when fragments of muscle were buried in damp earth, the muscle and surrounding earth, after some weeks, were found to be swarming with nematoids of this type. In the spring of 1866 (before all such work was interrupted by other avocations) I chanced to make one tentative experiment of this sort, with the following results:—Some earth was taken of a kind which I had often examined before without its yielding any nematoids, and a small fragment of uncooked mutton, about the size of the tip of my finger, was buried therein. Portions of this mutton were also previously examined with the microscope without revealing the presence of any nematoids. In the course of a few weeks, to my surprise, the earth and surface of the muscle were found to be swarming with minute nematoids such as I had never seen before, and which, if not belonging to the genus *Pelodera*, were some closely allied forms. In regard to this latter point I am not quite certain, as I seem to have preserved no specimens, and am also unable to find any drawings of them. This observation inspired me with the desire of making a set of differential experiments under stricter conditions, using different kinds and differently treated earth on the one hand, and different varieties of muscle on the other—that is, from fish, bird, and mammal. Unfortunately, however, from the pressure of other work, I have since found no leisure for following up such enquiries.

The question whether the "free nematoids" generally are parasitic in any stage of their existence is one which is of considerable interest. And the answer that should be given to it is, I think, that such a parasitic stage is no necessary part of their life-history, though many of the forms may and do become parasitic in a more or less accidental manner—partly infecting various portions of plants, and partly, perhaps, different tissues or organs of various animals.

That they are not necessarily parasitic is clearly shown by the fact that the several kinds are to be met with in all stages of growth and development in their respective external media.

But that they are occasionally parasitic is no less obvious, and especially in plants of various kinds. Thus it has been long known that a disease of wheat (commonly called "purples," or "corn-cockle") was associated with a minute nematoid (the so-called *Vibrio tritici*), and this organism I some years ago ascertained to belong to the genus *Tylenchus*. After referring to the mode of infection of the wheat as discovered by Davaine (*i.e.*, owing to the young nematoids passing from the earth in which wheat is growing, getting between the sheaths of its leaves, and proceeding thence onwards to the immature germens), I have elsewhere added*—"In several grasses I have found different species of these free "nematodes lying between the inner sheaths of the leaves near the "bottom of the culm. In *Festuca elatior* I met with no less than "five species in this situation, belonging to the genera *Dorylaimus*, "*Mononchus*, and *Plectus*; and in the stalks of wheat and oats removed "from stubble-fields I have frequently found specimens either of these "genera or of *Rhabditis*, *Aphelenchus*, or *Cephalobus*. In addition to a "malady of oats and maize, similar to that of wheat, and said to be pro-"duced by the same animal, Steinbuck,† nearly a century ago, recognised

* Loc. cit. p. 88.

† Naturforsch, xxviij. s. 233, tab. v.

" a disease somewhat similar to the 'purples,' in two of the bent-grasses " (*Agrostis*); and from the frequent presence of these nematoids in " the situation named, I suspect such diseases of grass will be found " more frequently, if specially looked after."

Kühn* long ago ascertained that a morbid condition of the flowers of the common teasel (*Dipsacus fullonum*) was occasioned by the presence of a number of these nematoids; and a somewhat similar condition has been more recently met with in connexion with the flowers of the common feverfew (*Pyrethrum parthenium*) by an author whose name I cannot now recall. Carter† discovered multitudes of these nematoids projecting from the conceptacles of some of the larger fungi of the genus *Zylaria* in Bombay; and Cobbold found specimens, which proved to be presentatives of the genera *Aphelenchus* and *Plectus*, very abundant in the decaying pulp of some pears‡.

A few years ago I ascertained that the nematoids which occurred not unfrequently in the diseased and nodose roots of certain cucumber plants in this country belonged to the genus *Tylenchus*. And last year I received an interesting pamphlet from Dr. Bancroft, of Brisbane, Australia, on certain "Diseases of Animals and Plants," from which I find that an almost similar disease is very common in Queensland, amongst the bananas. From the merely rough figures there given of the young worms, it seems evident that these nodose roots of the banana are also infested by nematoids of the genus *Tylenchus*. The parent females become so enormously distended with young that they assume somewhat the shape of a Florence oil-flask, so that Dr. Bancroft speaks of them as "flask-worms." § He makes the following important statements in regard to the occurrence of such worms in the roots of bananas and of other plants:—"The root of *Musa maculata* breaks off " where the worm colony has been located; the sugar banana is more " tolerant of the parasite, and does not break off. This latter is the " species of Musa which produces the fruit now sold in Brisbane. On " examining further for the flask worm, I find scarcely any roots of " cultivated plants free from them. It is common in most of the roots " of culinary vegetables, and many weeds, particularly *Sida retusa*; " indeed, I have found the roots of Sida covered with these excrescences " here exhibited in waste ground never under cultivation, hundreds of " yards from gardens. I am satisfied the flask-worm is doing much " injury in gardens." Dr. Bancroft also speaks of having found the flask-worm in diseased vine roots.‖

In regard to the occurrence of such organisms as parasites of animals, we as yet know extremely little. Still, some hints have been thrown out upon this subject which should not be overlooked.

Dujardin included many different forms of free nematoids under the one genus *Rhabditis*.¶ Speaking of his *R. terricola* he says: "Cet " helminthe si remarquable par sa structure, ne l'est pas moins par son

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* Zeitsch. fur Wissen. Zoolog., 1857, t. ix., p. 189.

† Trans. of Med. and Phys. Soc. of Bombay, 1861, App. p. 1.

‡ *See* Monograph of the Anguill. (Trans. of Linn. Soc.), p. 124.

§ Three or four years ago I recognised the same peculiarities in regard to the Tylenchi found in cucumber roots, and further, that no males were to be seen. In this latter respect, and in regard to the great distension of the females with embryo nematoids, we may be reminded of what is known concerning the guinea-worm, only in this latter case the increase in size comes to be truly enormous.

‖ And I believe the cabbage plant in this country is known to be affected at times with a disease similar to that which is met with among cucumbers.

¶ On this subject see Trans. of Linn. Soc., vol. xxv., p. 129.

" habitation dans la terre humide et parmi les mousses, où il peut subir " une desiccation complète sans périr, et d'où il est entrainé par la pluie " dans les fossés et les rivières. Il passe ensuite comme nourriture " dans l'intestin des limaces, et de là dans l'intestin de la grenouille " rousse, qui devore ces mollusques; ou, bien, il est avalé dans les eaux " par les gastérostés et divers petits poissons."

These certainly are modes by which higher animals might become the hosts of free nematoids, and some of such nematoids may have the power of living and multiplying in one or other of these accidental situations.

Whether the minute nematoids discovered three or four years since in the dejections of persons suffering from endemic diarrhœa in Cochin China, and recognized also during the present year in the dejections of workmen (suffering from profound anæmia) who had been engaged in the excavation of the Mont Cenis tunnel, are, as some have asserted, members of the genus *Rhabditis*, must for the present be regarded as somewhat doubtful. I have seen no good drawings of them, nor have I yet had an opportunity of examining any of the specimens.

It has, however, been suggested by H. J. Carter* that certain young free nematoids may make their way into the sub-cutaneous tissues of man, perhaps by way of the perspiratory ducts, and there develop into the human parasites known as guinea-worms. Certain it is that such parasites are found almost solely in regions of the body exposed to external contamination by water and what it bears. In four-fifths or more of the whole number of recorded cases, guinea-worms have been situated in the lower extremities and below the knees of persons who are in the habit of going about with bare legs and feet. Some notable exceptions in regard to regions implicated have been referred to by Bruce in the case of certain Indian water-carriers. These men bear water on their backs in leather bags or skins, and it is found that they are not unfrequently affected with guinea-worms in an altogether unusual situation, viz., about the back and loins. All such facts are quite inexplicable by the hypothesis of Fedschenko, supported by Dr. Cobbold†, to the effect that the young of the guinea-worm penetrate the bodies of certain entomostraca, such as *Cyclops*, and are thereafter swallowed with their hosts by some of those persons who drink unfiltered water. The fact that in certain experimental observations Fedschenko saw the young of the guinea-worm bore their way into the abdominal cavity of Cyclops may be perfectly true, and yet his hypothesis as to the way in which human beings become infected with guinea-worms may be wholly incorrect. It supposes that of the members of one or more broods of young nematoids produced within the alimentary canal of their human hosts only one or a few worms for the most part find their way to the sub-cutaneous tissue, and that almost invariably and solely to a region of the body open to the risk of direct contamination from without.‡

An alternation of free and parasitic generations of nematoids of a very peculiar kind is, indeed, believed by Leuckart and Mecznikow, to obtain in some cases.§ Thus *Ascaris nigrovenosa*, often to be found in the lung of the frog, is said to exist also alternately as a free nematoid which, from the figures given by Leuckart (*Die Menslichen Parasiten*,

* Annals of Nat. Hist., 3rd ser., vol. i., 1858, p. 410, and vol. iv. 1859, pp. 28, 98.

† "Parasites; a Treatise on the Entozoa of Man and Animals," 1879, p. 233.

‡ The writer has considered some of the questions relating to guinea-worm infection and development in Trans. of Linn. Soc., vol. xxiv. (1863), pp. 106, 122–128.

§ *See* Bull. de l'Acad. Roy. des Sc. Belg., No. 8; 1866, p. 208.

Bd. II.; figs. 106–110), would seem to belong, if not to the genus *Rhabditis*, to some very closely related type. But, that the young of any such creatures should develop into a totally different form of nematoid, worthy by its different structural characters of being called an *Ascaris*, is a statement which I find it hard to believe, notwithstanding the authority of those by whom it is made.

It is worthy of note that all free nematoids cannot resist complete desiccation as some have supposed, nor is this potentiality possessed by those creatures now included under the genus *Rhabditis*, as might be imagined from Dujardin's previously quoted statement. After having made special observations for the purpose of throwing light upon this subject, I have elsewhere said*:—"The different members of this "group vary much as to their tenacity of life. As a rule they are "frail and delicate, and do not recover even after a slight desiccation of "five or six minutes, thus differing remarkably from what I have at "present observed with members of the four land and freshwater "genera, *Tylenchus Plectus*, *Aphelenchus*, and *Cephalobus*. With "all these there is a remarkable tenacity of life and power of recovery "after what seems to be complete desiccation."

If future observations should confirm the view entertained by Mr. Power and Dr. Cory that such nematoids as were found in the body of the boy Pierce caused the malady from which he died, and were causes likewise of the various other cases of febrile illness that occurred in epidemic fashion amongst the residents on board the "Cornwall," the importance of their discovery will be increased rather than diminished by the fact that the nematoid in question is not, as they at first naturally enough supposed, *Trichina spiralis*, but some hitherto unknown form producing a previously unrecognised disease, whose clinical affinities are almost as strong in the direction of enteric fever as they are in that of trichiniasis. In this case we shall, in future, have to distinguish between two distinct "fleshworm" diseases in the human subject, the one caused by the Trichina fleshworm, and the other by the Pelodera fleshworm.†

The mode in which worms of the latter class, or their allies, gain entry to the body would still remain to be determined. Some of such modes have been already sufficiently hinted at, so that it is only necessary now to name the eating of raw fruits or vegetables contaminated with such organisms or with minute slugs that may have swallowed them, in addition to the drinking of unfiltered and unboiled water which may for some time have been stored in tanks or cisterns containing much organic sediment, as possible modes by which dwellers in towns or other communities might be infected. The young or the ova of particular free nematoids might, from time to time (that is, at particular seasons or under particular conditions), chance to be brought to such reservoirs in unusual numbers, and they might there find conditions favourable for their still further multiplication. And if disease, in man, does not often result from such causes, this is probably to be ascribed to the general inaptitude of these free nematoids to take on this kind of accidental parasitic existence rather than to the rarity with which they or their ova are able to gain access to the human alimentary canal.

June 17, 1880.

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* Loc. cit., p. 85.

† Peloderiasis would also be a name by which the new affection might be distinguished from trichiniasis.

No 8.

App. A. No. 8.

On Enteric Fever at Red Hill and Caterham, by Dr. Thorne Thorne.

On an Extensive Epidemic of Enteric Fever at Red Hill, Caterham, and other Adjoining Places, by Dr. Thorne Thorne.

On February 4th, 1879, the Local Government Board received a communication from Dr. H. C. Hilliard, of Caterham, to the effect that within the previous 10 days 25 cases of enteric fever had come under his notice in that place. The outbreak was explained to have been sudden in its occurrence, and, as the result of some inquiry which he had made, Dr. Hilliard stated his belief that the disease had been conveyed by means of the water supplied by the Caterham Waterworks Company. Under these circumstances I, on the same day, received instructions to make immediate inquiry into the causes of the outbreak. This inquiry was commenced on February 5th, and it was continued at intervals until early in the following month.

On reaching Caterham, I found that the other medical practitioners in the place had also been called in to a considerable number of cases of the same disease. The first person attacked had sickened on January 19th; a second attack occurred on January 20th, two more on the 23rd, three on the 24th, and from then up to February 2nd, to which date information was at first limited, fresh attacks in fresh houses had occurred day by day, the total number of houses affected being 35, and the total number of sick being 47. The daily number of attacks which came under medical treatment had varied from one on January 19th, 20th, and 30th, to ten on February 1st. This group of 47 cases, included as they were within a fortnight, was at once made the subject of inquiry.

Caterham is situated in the rural sanitary district of Godstone, and it has a population of about 5,800. One part of it, which will in this Report be called Lower Caterham, occupies a position near to the southern extremity or head of the Caterham Valley, a valley bounded by chalk hills, extending north and south for a distance of about 5 miles, and opening out at Caterham Junction about half way between Caterham and Croydon. The houses in Lower Caterham consist mainly of superior villa residences, many of which have been built within recent years. The other part, or Upper Caterham, lies at a higher altitude at the southern end of the range which bounds the valley on the west. Here also are a number of villas, the old village of Caterham, one of the asylums belonging to the Metropolitan District Asylums Board, and the barracks of H.M.'s Foot Guards.

The cases of enteric fever above referred to, and which had occurred in the fortnight ending February 2nd, were spread over a very wide area, some in Upper, some in Lower Caterham, extending to the extreme outskirts of those places. The houses attacked belonged to no special class, both rich and poor having suffered. It was at once apparent that the disease could not have been conveyed to the affected houses by means of any general system of sewers common to the district, for by far the majority of the houses were found to drain into separate cesspools excavated into the chalk; some of these cesspools, as also the house drains, being efficiently ventilated, others having no such ventilation. Some of the houses affected had no artificial means of drainage, the liquid refuse being thrown on to gardens adjoining them. There was also no possibility that there had been any common cause of infection in connexion with the prevailing means of excrement disposal, because there was nothing in common with regard to such disposal. Most of the houses in which the disease had appeared were provided with water-

closets, a considerable number of which were apparently well trapped and ventilated. Others had closets which were fitted with a trap and pan and emptied into cesspools, and though these closets were at times a source of nuisance owing to the absence of any flushing apparatus, yet they were all situated out of doors. Others again were provided with common privies and a few with earth-closets. The possibility of infection having been communicated by means of a milk-supply was next inquired into, and it was ascertained that 33 of the houses affected received their milk from at least five different and completely independent dairies, and that, at the remaining two, private cows were kept. It was also evident that personal infection could not in any way have led to the outbreak.

Further, there was no history of any recent prevalence of enteric fever in Caterham. It was stated that the locality for some years past had been remarkably free from the disease, and during the twelve months preceding the outbreak only one isolated case could be heard of. This is believed to have been an imported case, and it terminated fatally on August 16th, 1878.

With regard to the water-supply, it was ascertained that out of a total of 558 houses in Caterham, 419 were provided with water from the mains of the Caterham Waterworks Company, the remaining 139 deriving their supply from local wells or from rainwater tanks and barrels. Of the 47 persons attacked during the fortnight January 19th —February 2nd, 45 resided in houses where the water of the Caterham Waterworks Company was in use, a circumstance which, having regard to the other points already adverted to indicated a likelihood that this water had been the means by which the infection had been conveyed. This view received confirmation when it was further ascertained that the two remaining patients, who resided on premises where private wells were in use, had, owing to the nature of their employment, not only been in the habit of spending the day at houses to which the Company's water was laid on, but had admittedly used this water.

I further ascertained from Dr. James Adam, Medical Superintendent of the Caterham Asylum, that no case had arisen in that establishment, where nearly 2,000 patients are under treatment; and also from Dr. James Magill, Coldstream Guards, that there had been a similar immunity from the disease amongst the 500 men under his charge in the Caterham Barracks. Both these establishments derived their entire water-supply from a well which belongs to the Metropolitan Asylums Board, and which is sunk to a depth of 462 feet into the chalk. The same has remained true up to the present time.

In the meantime, however, I received information from several sources, notably from Mr. E. L. Jacob, Medical Officer of Health for the combined sanitary districts of Surrey, to the effect that enteric fever was also epidemic in Red Hill.

At Red Hill, inquiries similar to those which I had made in Caterham, had been undertaken by Mr. Jacob, and to him I am indebted for the greater part of the detailed information as to the cause of the epidemic in that locality.

Red Hill, including Earlswood village, the Earlswood Asylum for Idiots, and a district known as Mead Vale, forms the eastern ward of the borough of Reigate, and it has an estimated population of 9,500. It is situated about eight miles by road from Caterham, and it differs from that place in several material respects. It lies on a different geological formation, namely, on the lower green sand; it is nearly throughout provided with a modern system of sewers, which is believed to be efficiently constructed and efficiently ventilated, and many of the

drains connecting the houses with the sewers are also ventilated. But, in regard of the epidemic of enteric fever, the two places presented certain remarkable points of similarity. In both the epidemic had commenced at about the same time. In Red Hill, the first two attacks were reported to have occurred on January the 20th; these were followed by 3 others on the 21st, 5 more on the 22nd, 12 on the 23rd, and at the expiration of the first fortnight, viz., by February 2nd, fresh attacks in fresh houses had every day occurred until the total number of houses affected was 96, and the number of patients had reached 132. The sewers, it is true, were common to the greater part of the district affected, but in this respect there were some striking exceptions, to which reference will be made, and so far as prior cases were concerned from which the infection could have been communicated by means of the sewers, there was evidence to the effect that Red Hill had been for at least eighteen months past quite free from the disease. The milk-supply of the houses affected was also ascertained by Mr. Jacob to have been derived from many sources having nothing in common, either amongst themselves or with the Caterham dairies. The water-supply for the greater part of Red Hill is, like that for Caterham, derived from the works of the Caterham Waterworks Company, and I found that, as the result of an early investigation of the epidemic, Mr. Jacob had, quite independently of either Dr. Hilliard or of myself, arrived at the conclusion that the spread of the disease was in all probability due to this water. Red Hill contains about 1,700 houses, and of these 924 had the Company's water laid on to them. Of the 96 houses affected during the first fortnight of the epidemic 91 habitually drew their water from the Company's mains, and the histories obtained with reference to the attacks in the remaining 5 were such as to confirm the impression that the Caterham Company's water had been the immediate cause of the epidemic.

Thus, at one of these houses, where ultimately three persons were attacked, the supply had, it was hitherto believed, been exclusively derived from a rain-water tank, but it was now ascertained that the Company's water was, in addition, procured surreptitiously; at another, for which there was apparently no water-supply, the Company's water was procured from a neighbour; and with regard to the remainder, the patients affected were not only persons who were employed where the Company's water was in use, but several of them had partaken of the water at their meals.

The following evidence, all tending in the same direction, was also during an early stage of the investigation communicated to me by Mr. Jacob.

Mead Vale is a locality within the eastern ward of Reigate borough. It contains about 160 houses, of which 140 are connected with the Red Hill sewers and 20 drain into cesspools; all, however, have their water-supply from local wells. Two attacks in two different houses had occurred in Mead Vale, but both persons were employed at the Earlswood Asylum for Idiots, where the Company's water was at the time in use. Of these two houses one had cesspool drainage.

In Red Hill itself is a group of 30 houses which are supplied with water from a well at the South-Eastern Railway Station. These houses are in close proximity to and immediately surrounded by others to which the Company's water is laid on; both groups being connected with the public system of sewers. In the 30 houses referred to, not a case of enteric fever had occurred, whereas from the houses around them, numerous cases were reported.

Reigate town also, which forms the western ward of the borough, and has a population of about 8,500, but which has a different water-

supply, entirely escaped, only two cases, which were undoubtedly imported from Red Hill, having occurred there.

Thus, all the facts ascertained in connexion with the course of this epidemic up to February 2nd afforded very strong presumption that it had been caused by the use of the Caterham Company's water. All that has arisen since that date goes to strengthen this view.

The Caterham Waterworks Company, at the date when this inquiry was commenced, had two deep wells, both sunk in the summit of the high-lying ground which forms the western boundary of the Caterham Valley near its southern extremity. These two wells are situated about 30 feet from each other; they first pass through about 140 feet of gravelly soil, consisting of Oldhaven pebble beds with a little clay and flints. At this depth the chalk is reached; it consists partly of soft and partly of hard chalk, the formation being about 310 feet in thickness. Below this is the upper green sand, having a depth of about 55 feet. For about 490 feet, both wells are several feet in diameter, and they are connected by three adits in the chalk. From the bottom of the two wells and from the lowest adit a series of borings have been made through the upper green sand down to the gault; an experimental boring, which has since become silted up, passing through the gault into the lower green sand. From these wells the water is pumped into a series of tank reservoirs, in which it is submitted to Clark's softening process. These reservoirs, some of which are of recent construction, freely communicate with each other, and from them the supply is delivered by gravitation.

During the past twelve months a third well and boring has been made for the Waterworks Company by the Diamond Rock Boring Company. This well is situated within 90 feet of the others, but it extends through the gault, which is here 343 feet in thickness, into the lower green sand below; this latter formation having been reached on February 22nd, 1879. Since 1861, and until the construction of the third boring was undertaken, the water supplied by the Caterham Company was held in high repute in the district to which it has been delivered, and the freedom from such diseases as enteric fever and diarrhœa which is stated to have been experienced in the district, has been to a great extent attributed, especially by Mr. Jacob, to the wholesome water-supply. Since the third well has been begun and prior to the epidemic under consideration, certain complaints were indeed made with regard to the water, but they were ascertained to be due, under circumstances to be hereafter explained, to conditions resulting from the recent boring operations, which had led to unavoidable turbidity in the supply and which had also prevented the process of softening from being regularly carried out. There is, however, no reason to believe that this unavoidable turbidity or temporary hardness of the water prejudicially affected those consuming it. In short, considering the deep sources of this water, and the previous history attaching to its use, it was by no means apparent how it could latterly have been the means of producing an extensive epidemic of enteric fever.

With a view to the elucidation of this point, it was first sought to ascertain whether any pollution of the water had taken place in the mains after it had left the Company's works, owing to intermissions in the service. This appeared at first to be possible, for not only was the occurrence of such intermissions reported to me, but during the course of this part of the investigation two waterclosets were found in Caterham, the water-supply for which was derived from service-pipes which passed direct from the main into the closet pans without the inter-

vention of cisterns, and were fitted with screw-down taps. The reported intermissions were, however, on investigation ascertained to have been temporary stoppages in the supply, caused by the freezing of the water in service-pipes, and I was assured by the Secretary to the Company that for at least many months past the Company's pipes had remained always full and under pressure. No intermissions, it was added, had occurred even during repairs, the service not only being a constant one, but the fixing of new service-pipes being in addition always effected under pressure whilst the mains are fully charged. Moreover, the physical circumstances of the two closets, improper as they were, did not appear such as would in fact have permitted entrance of any foulness from them into the water pipes. No such dangerous services to waterclosets were found in any other portion of the district supplied by the Company, and in the two cases above referred to they have since been done away with.

I next made inquiry with a view of ascertaining whether any accidental contamination of water in the reservoirs, or in the mains, could have resulted from any irregularities in the supply during the recent boring operations or otherwise. I found that during the course of these works, and owing to the turbidity which has been referred to, the Caterham Waterworks Company had on several occasions procured an additional supply, which was pumped into their mains from two sources; the one, the deep chalk well belonging to the Caterham Asylum, the other, a well at Kenley, a place situated in the Caterham Valley between 3 and 4 miles to the north of Caterham. This latter well, which is also sunk in the chalk to a depth of 150 feet, belongs to the Kenley Waterworks Company, and it supplies the rapidly increasing groups of villa residences at Kenley and in the immediate neighbourhood.

With regard to the water derived from the well at the Caterham Asylum, it must be noted that it ceased on December 30th, 1878, and was not resumed until January 30th and 31st, 1879, when a further supply was delivered into the Company's mains. Now if the water delivered by means of the Caterham Company's mains was the vehicle by which infection was conveyed, that infection must have been first distributed through them about 10 or 14 days prior to the occurrence of the first cases on January 19th and 20th, that is to say, between January 5th and 9th; and since no water from the asylum well was for many days prior to or subsequent to that period being delivered to the Company, it cannot have been concerned in the distribution of the infection. The evidence, too, already quoted as to the immunity of the asylum and the barracks from the disease, renders any further consideration of this water as a possible source of the infection quite unnecessary.

The supply from the Kenley Waterworks Company was delivered continuously every night into the Caterham Company's mains from November 26th, 1878, to January 3rd, 1879; it was resumed again from January 5th to the 10th, after which it was discontinued until the nights of January 14th and 15th; this supply finally ceasing on the morning of January 16th. This water was therefore pumped into the Caterham Company's mains for the period (January 5th to 9th) above referred to as that during which the distribution of infection must have commenced. Though the Kenley water, if it had had mischievous quality, would thus have had opportunity of doing mischief, there is absolutely nothing to suggest that it possessed such quality. On the contrary Kenley itself, which never ceased to be supplied from its own well, remained quite free from enteric fever during the whole course of the epidemic. Further, I was informed that there are good engineering

grounds for believing that little or none of the Kenley water ever could have reached Red Hill, and it can therefore have had no concern in the fever there.

Many other points relating to the method of distribution of the Caterham Company's water were inquired into, but none led to any explanation of the circumstances of the epidemic.

As the Board are aware, suggestions of danger to the water in the deep wells of this chalk district, due to the prevalence of a system of cesspool drainage, have from time to time been made, and owing to this, as also to local representations, special inquiry was made as to the possibility of the soakage of filth from the surface soil around either of the Caterham Company's wells. Both the wells which were in use prior to the epidemic are steined with brickwork down to the level of the chalk. Not only is this brickwork not absolutely water-tight, but there was ascertained to be a somewhat free communication of water between the new boring and the other wells, and hence both the Company's premises and some neighbouring ones were examined, with a view of ascertaining whether any means existed favouring the pollution of the water in the method indicated. On the Company's premises a cesspool was found, but it was cemented and evidently water-tight. This cesspool has since been done away with. A similar one is stated to have been found some ten months earlier, and this was also at the time abolished, the soil for a considerable distance around being at the same time cut away. The only other cesspools in the vicinity of the wells, are the following: 1st, One situated at a house about 1,100 feet to the south of the wells. This is a cemented tank, the contents of which are pumped on to the land. 2nd, One which is situated at some stables about 360 feet to the north of the two older wells, and about 270 feet in the same direction from the new bore: but this was originally constructed as a rain-water tank, and it is known to be water-tight. 3rd, Another on the same premises, and situated about 570 feet from the older wells, and about 480 feet from the new bore. This cesspool is about 6 feet in diameter and 15 feet in depth; it is stated to be dug in a clayey soil, and its contents overflow on to some meadow land. It is not cemented and it is believed to be "nearly" water-tight. Danger of soakage into the Company's wells from this cesspool is probably very remote, if at all possible.

In estimating the risk, regard has been had to several points which I propose to note here, not so much because of any danger believed to have resulted from this cesspool up to the present date, but mainly by way of caution as to the construction of cesspools in this locality in the future.

In the first place the clayey nature of the soil is relied on to prevent soakage. From a description of the soil in this neighbourhood which I have received from Mr. W. Whitaker of Her Majesty's Geological Survey, it appears that the hill in which the waterworks and the above-named premises are situated is an outlier of pebble-gravel (belonging to the Oldhaven Beds), resting irregularly on the chalk, and flanked on the north-west and north by an irregular deposit of pebbly loam, of much later date, which masks the junction of the Oldhaven Beds and the Chalk. The Oldhaven Beds at this point consist to a great extent of a deposit of well-rounded flint pebbles in a sandy matrix, but in places are irregular masses of angular flints in a loamy matrix, and it is highly probable that the cesspool in question has by a fortunate accident been sunk in one of the latter places. Not only can such a soil not be relied on elsewhere in this vicinity, but there is no security that the chalk itself may not be met with, so irregular is its surface. Indeed, according to Mr. Whitaker, it appears that on one side of one of the Company's wells it is met with at a depth of 90 feet, on the other at 140 feet, and that it may elsewhere come much nearer to the surface.—Again, the dip of the strata at this point is northwards at an

angle of about five degrees, and both the surface-slope and the flow of underground water are stated to be in the same direction. Hence it has been assumed by some, that the danger of soakage from a cesspool situated at a reasonable distance to the north of the wells need not receive serious consideration. This northward flow of the underground water is, however, only one of the elements to be considered. Within the cones from which these wells are supplied the flow of the water is, owing to the existence of fissures in the chalk, if not, also to other causes, at times in other directions. Thus, during the boring operations which have been carried on by the Diamond Rock Boring Co., it was found necessary to pump into the bore itself about 3,000 gallons of water every hour, to facilitate the process of boring, and special arrangements had been made to receive the water as it rose from the bore into tanks, from whence after subsidence it might again be utilised for the same purpose. Shortly after these arrangements had been carried out, however, it was found that although this bore is situated to the north of the other wells the whole of this water escaped through a fissure which had been reached in the chalk, and which is believed to be about 14 feet in length, and through this fissure the water found its way in a southerly direction through the intervening 90 feet into the other wells. This it was that led to the turbidity in the supply which had been complained of. Somewhat similar occurrences have been observed with regard to water which has been allowed to run to waste on the surface. On one occasion the waste water was pumped into a hollow spot, about 170 feet to the east of the wells; here it disappeared, but it was soon ascertained to be returning into the wells at a depth of 420 feet, that is about the level of the "upper spring" supplying them, and it was found necessary to discontinue the process. On another occasion the waste water was led by means of a trench to a spot on the slope of the hill to the east of the works, and about 320 feet from the wells. From this point also it found its way back to the wells. Under the circumstances narrated it is clear that, although there are no grounds for believing that danger has actually arisen from the existence of any cesspools near the Company's wells, yet a system which allows, within the drainage area of a well, of soakage from cesspools, is obviously one calling for immediate remedy on the part of the Sanitary Authority.

Circumstances had, however, in the meantime come to my notice which had led to the inquiry with reference to any possible contamination of this water-supply being extended in another direction.

During the latter part of 1878 and the beginning of 1879 the Caterham Waterworks Company constructed an adit from one of their old wells up to the new bore which was then being sunk. This adit is situated in the chalk at a depth of 455 feet, it measures 6 feet by 4 feet, and is 90 feet in length. A number of men were employed in this work, some of them being in the well below, others on the surface.

Inquiry was made as to whether any of these men had ailed whilst at their work, and it was ascertained that one of them who left work some time in January was believed to have been ill, although no inquiries had been made concerning him since he quitted the works. This man I sought out, Mr. Jacob accompanying me on the occasion. At first his wife only could be found, but subsequently an interview was had with the man himself, and from the statement of the two the following history was obtained:—

J. K., aged 32 years, the man in question, resides in Caterham, and he entered the service of the Caterham Waterworks Company on October 25th, 1878, being employed as a labourer in the construction of the new adit referred to. The position assigned to him was that of "loading man," he being employed in the adit below in attaching to a rope, let down from above, the buckets by which the excavated chalk was raised to the surface, and in again receiving those buckets when lowered full of bricks and cement, required for the work in progress. Owing to a temporary inability of the pumps to keep down the water below the level of the adit, work was discontinued on December 14th,

when J. K. was discharged, and it was not until December 29th that he was again sent for to resume the place he had formerly occupied. When he returned he was in perfect health, but "in about a week," *i.e.*, about January 5th, 1879, he felt himself ailing. His symptoms, which, according to his statement, steadily increased, were at first loss of appetite, recurring attacks of shivering alternating with a feeling of heat, great pains in the limbs, which he attributed to rheumatism, but which instead of being confined to any of the joints, were described both by himself and his wife, as an "aching all over," and diarrhœa. As the symptoms became aggravated, he was so exhausted during his work and became so "giddified" that he was more than once drawn to the surface, and immediately on his return home he was compelled to go to bed. More than once his wife noticed that he was "light-headed" in his sleep. All this while the diarrhœa continued, the man making a great effort to remain at his work, because, as he explained, he had had no employment between the 14th and the 29th of the previous month.

With reference to this man's diarrhœa it is necessary to make the following explanation:—Both from his own statement, and from that of others, it appears that all the men who worked in the adit were expected to make such preparation before descending the well, that no occasion should exist for relieving themselves below; but should such necessity ever arise, and should there be at such a time any difficulty or delay in their being drawn to the surface, the buckets which were regularly being raised to the surface were to be used for that purpose. J. K. states that he strictly complied with these regulations before descending, but that notwithstanding all his efforts, the purging under which he was labouring was such that he was compelled to evacuate whilst in the adit "at least two or three times" during each shift, the shift lasting apparently from 8 to 12 hours each according to circumstances. Indeed, as time went on, the man's diarrhœa must have been considerable, for besides the attacks which came on whilst in the adit, he almost invariably suffered from it, before descending, immediately after ascending, and also at his own house. So matters continued until January 20th, when work was again suspended for two days on account of a rise in the water level. But during the night of the 21st he was so much worse that he was unable to rise next morning. According to his wife's statement he found he could not stand when he got up, and returning to his bed suffered from "shivering down the back," "aching and exhaustion," and later on severe abdominal pain came on, which compelled him to lie with his knees drawn up: he was also "burning hot." This pain was looked upon as "cramp," and was alleviated by linseed meal poultices which were applied by his wife. The more severe symptoms, including the diarrhœa, having subsided, he was two days afterwards able to get up for a while, and from this time convalescence appears to have set in. No medical advice was sought, mainly, as he explained to me, owing to his straitened circumstances. When I saw him, on February the 8th, he had the aspect of a man who had recently suffered from some acute disease; he was still very weak, and it was obvious that he had greatly lost flesh.

As the successive points in this history were learned, more and more resemblance appeared to a mild case of enteric fever, until, after the whole of the above story had been learned, and after the man himself had been seen, the diagnosis of this fever could be made with a considerable degree of certainty. Then inquiry as to the man's movements at the period when the cause of his own fever must have been operative, brought out the fact that he had spent December 25th and 26th, 1878, at Croydon, so that he had had opportunity beyond what Caterham then afforded for getting the fever.

On the morning following my interview with J. K., I proceeded to Croydon, with a view of ascertaining whether any clue could then be obtained with regard to the possibility of the man having in any way been exposed to the poison of enteric fever on December 25th or 26th; the only accurate history of his movements during those two days relating to that part of them actually spent in Croydon. It appears that on the days in question there had been a gathering of his friends at the house of a relation. The information which I obtained there was, however, entirely negative; at the same time it was evidently incomplete and otherwise unsatisfactory. For, after the lengthened examination to which J. K. had been subjected on the previous day, he apparently apprehended the object I had in view, and during the same evening, some one had gone over from Caterham and had evidently informed the relative at Croydon of my intended visit. As the result of this, certain information relating to the residence and antecedents of some of the visitors at the gathering, and desirable for the completeness of my investigation, was practically refused me. In making inquiry as to the possibility of any local source of infection, I received the assistance of Dr. Philpot, Medical Officer of Health for Croydon. The only sanitary defect, with regard to the premises themselves, was the existence out of doors of a pan-closet, trapped and connected with the public sewer, but not provided with proper means of flushing. It is, however, noteworthy, that at one of the small group of cottages in the side street in which the house in question was situated, it was learnt that during the previous October and November there had been a case of "low fever" in a child, and that shortly before Christmas the child's father had suffered from "acute diarrhœa."

I have now no hesitation in taking it as a fact that a man ill of enteric fever from January 5th to the end of the month was occupied during the first fortnight of that period at work in the well of the Caterham Waterworks Company. The fact, it will be observed, is not inferred from any consequences of it, but simply from what was seen and heard of the particular individual.

But now let us see what those consequences would have been. If this man's stools could by any means have found their way into the water of the well in which he had been working, and being enteric fever stools could thus have led to the development of the poison of that disease in the well, the effect on the water consumers ought to have been noticed within from about 10 to 14 days after the date when the diarrhœa first came on. And this, in effect, is precisely what did take place, the epidemic having commenced on January 19th and 20th in Caterham and Red Hill respectively. This remarkable concurrence of dates led to a more detailed inquiry as to the course of the man's diarrhœa whilst working in the adit. He admitted that the purging was very copious, in short that it "ran from" him; indeed, when at home, he was, because of the suddenness of its onset, unable to resort to the closet. He further admitted that owing to his frequent use of the bucket whilst at work, complaints were made by his fellow workmen on the surface; but he stoutly denied that he had ever been so pressed by necessity, or so influenced by those complaints, as to relieve himself in the adit without waiting for a bucket. But even accepting his denial, there were undoubted means by which his evacuations could have found their way into the water. According to his statement, the bucket was used as a closet when it was empty, when half full, and when full; he added, however, that when it was full he first took some of the chalk out and subsequently replaced it. During an earlier stage of my inquiry I had occasion to descend one of these wells, and I noticed that any article let down by a rope, by its oscillations to and fro, came into

constant and somewhat violent contact with the walls of the wells, and on inquiry of J. K., whether the same did not take place with the bucket, he admitted not only that this was so, but that some of its contents frequently fell over a stage into the water below. On further inquiry, he added that some portions of his evacuations probably did so also. And he further stated that the looseness of his bowels was such that the bucket itself must almost of necessity have been stained with them. This bucket, which was merely emptied out above, then received, as already explained, materials which were used in the construction of the works below. Here then were the stools of an enteric-fever patient, from about January 5th onwards getting into the Caterham Company's water and distributed with that water to the district served by the Company.

Now we know from ample experience that enteric fever is produced, and produced with the maximum of certainty, when the specific evacuations of that disease are consumed by a population. Again, it is a matter of experience that where enteric fever has been conveyed through water, some fortnight has to elapse between the distribution of the water and the occurrence of the disease among the community served by it. But a fortnight after January 5th is the very day when the first case of fever occurred, and during the fortnight following upon the period January 5th to 19th, *i.e.*, from January 19th to February 2nd, the disease became widely spread throughout Caterham and Red Hill; the distribution of the fever being limited, as we have already seen, to houses supplied with the water of the Caterham Company. There can, I think be no doubt but that we have in the man J. K. the cause of the disease which followed.

Hitherto, however, I have only dealt with the course of the epidemic up to February 2nd, namely, during the first fortnight of its progress, and I have only referred to it in so far as it related to Caterham and to the town of Red Hill. With regard to these two places no diminution in the epidemic was apparent until about the 11th and 12th of February. Up to about the 20th February, the disease continued exclusively to attack persons residing in houses to which the Caterham Company's water was laid on, or it selected from amongst those who habitually used another supply, those who had partaken of the Company's water occasionally, either whilst at their work or during a visit to houses to which it was laid on. After this time, however, the disease became, as might have been predicted, somewhat more diffused, and several of the later cases occurred in houses where there had been previous ones. Up to the end of February the total number of attacks in Caterham and Red Hill, concerning which information had been obtained, and including 13 cases in which the dates of attack could not with any accuracy be ascertained, was 305; of these, 224, of which 9 terminated fatally, occurred in the town of Red Hill, and 81, including 5 fatal cases, at Caterham.

The area of the Caterham Company's water service, however, includes other places than those hitherto named, and inquiry has been made in these places as to circumstances that should confirm or disprove the conclusions thus far arrived at. The information thus obtained, though not all as intelligible as that relating to Caterham and Red Hill, was, however, found to be in harmony with it when all the facts were ascertained.

The Caterham water was, together with another supply, used at the Earlswood Asylum for Idiots, this establishment standing in an isolated part of the eastern ward of the borough of Reigate. In this establishment the first case appeared on January 28th, and between this date and February

18th, 38* persons in all were attacked, 33 being patients and 5 servants. Five fatal cases occurred amongst the patients. Dr. Grabham, the medical superintendent, informs me that having noticed that the attacks were limited to those who were somewhat habitual water consumers, and apparently influenced by the turbidity which the Caterham Company's water had presented for some time past, he cut off that supply on February 8th, and limited the establishment entirely to a supply derived from local springs. The last attack in the asylum took place on February 21st, that is within a fortnight after the use of the Company's water was discontinued.

Between Caterham and Red Hill, and in the course of the main leading from the former to the latter place, the same water is supplied to a few houses in Godstone and Bletchingley, both in the rural sanitary district of Godstone, and to a few in Nutfield, within the rural sanitary district of Reigate.

Out of 622 houses in those three villages, 96 were provided with the Company's water, and in 9† out of the 96, fever prevailed. Of these 9, six were in Nutfield. No cases occurred in Godstone.

One more locality is supplied with this Company's water, namely, Warlingham, which lies about three miles to the north of Caterham, between that place and Kenley. One part of Warlingham, which is known as Whiteleaf, is a hamlet situated at the foot of the chalk hills, bounding the eastern side of the Caterham Valley; the other part lies on the hills and is known as Warlingham village. The population of Warlingham was 773 in 1871, and the Caterham water is laid on to 156 houses, viz., 102 in Warlingham village, and 54 in Whiteleaf. Throughout the course of the epidemic only one person is believed to have suffered from enteric fever in Warlingham, the attack occurring in a child 9 years of age. It is known that the Caterham Company's water was regularly drunk by this child at his home, but since he attended a day school in Caterham to which the Company's water was also laid on, it becomes impossible to speak with authority as to where he contracted the disease.

In view of recent investigations into the intimate pathology of the infectious fevers, the contagion of enteric fever itself must probably be regarded as particulate, and when contained in water it must probably be viewed as held in mechanical suspension, rather than distributed throughout it after the manner of a chemical solution. Under these circumstances its distribution throughout a large body of water would probably be not wholly regular, and its passage into and through mains receiving the water might not be uniform, either in point of time or in the direction taken.

So far, however, there is nothing in the case of the Earlswood Asylum, or of the intermediary villages, but what is confirmatory of the experience of Caterham and Red Hill. The actual rates of houses attacked are not identical in the several places; but it could hardly be expected that they should be, for incidence of disease on any particular house must have been determined by a multitude of minor circumstances; and any small group of houses, whether in Caterham or Red Hill, or in some outlying village, had a chance of being attacked to a greater or less degree than the average of houses supplied with the Company's water. As one of the many circumstances to which I refer, I may mention frost

* Two of the cases amongst the servants are, in the Table appended, included in the attacks at Red Hill. The persons attacked had their houses in that place and were attended there.

† This number does not include several houses in which imported cases sickened with the disease.

as affecting the water-services of houses. During the time that the Caterham water was mischievous there were many frosts, so much so that the notion of intermission arose. At Godstone this was specially noticed. The inequality of incidence, more at Nutfield, less at Godstone, then, is only what might have been anticipated.

But there remains the case of Warlingham, which has appeared at a first view more difficult of explanation. This is a village somewhat largely served by the Caterham Waterworks Company, no less than 156 houses being supplied from that Company's mains. Amongst 156 houses, we should expect at the Caterham and Red Hill rate to find some 12 houses attacked. But beyond the single case referred to, nothing was heard of the disease in the village. Why was this?

In proceeding to consider the answer to this question, let the special situation of Warlingham, in regard to its water mains, be first observed. The village of Warlingham lies about three miles from the Caterham Water Company's works, and for the greater part of that distance it has a special three-inch main to itself and to a few houses between Caterham and Warlingham. The capacity of this main and of its branches is somewhat over 12,200 gallons. What if this main can have been charged with a different water and not with the water from the Caterham Company's wells?

Now, in a former part of this Report, I have told of the Kenley water being used to supplement the Caterham Water Company's supply during certain portions of the time when the Caterham wells were, for reasons named, furnishing a diminished and—as the result showed—an injurious supply. The Kenley water was, however, not mixed equably with the Caterham water. It was pumped into the Caterham Company's mains at one particular point. *And that point was at the extreme further end of the Warlingham branch of the system.* Before the Kenley water could get to the Caterham Company's reservoirs, it had to fill the whole of the three-inch Warlingham main and its branches, besides one other larger main near to Caterham. Every night an amount of Kenley water, which is variously estimated, but which at the lowest estimate averaged between 13,000 and 14,000 gallons, was pumped into this Warlingham system towards Caterham, and every day some 12,000 gallons of this Kenley water would have to be consumed at Warlingham before Warlingham could obtain a drop of water derived from the wells at Caterham. While this was going on there was for each house at Warlingham a supply of some 70 gallons and more of Kenley water to be got rid of before any of the Caterham water could be procured.

It is true that on particular nights, and those during the time that the Caterham well water had dangerous properties, this pumping of Kenley water was not going on. From January 11th to January 14th, more or less of Caterham water must have found its way in the direction of Warlingham. Whether it actually got there before it was met and driven back again by the Kenley supply, which on the 14th was again introduced into the end of the Warlingham main, or whether the Kenley water pumped in before the 11th was all that the village ever received during that interval, is more than can be decided with any degree of certainty.

For my own part I am quite prepared to believe the latter of these alternatives, the more so as I ascertained that the consumption of water in Warlingham was, at the special period referred to, as also during the greater part of January, at its minimum. For, owing to the exposed position of the service-pipes and taps, and to the hard frosts which then prevailed, hardly any water at all had in several parts of the village been obtained from the mains for several days; the inhabitants resorting to rain-water which is largely stored in the village. And even when

the Company's supply was procurable it was, owing to the same cause, a mere "dribble."* This, however, is abundantly clear: the delivery of the harmless Kenley water to the village of Warlingham was so special and was continued during so large a part of the time when other places were being injuriously affected by the Caterham water, that the chances of suffering from any injurious influence carried by the latter water must have been out of all proportion less in the case of Warlingham, than in the case of places that got the bulk of their supply from the Caterham wells. And I am inclined to believe that, in the single case of enteric fever reported from Warlingham, the opportunity for infection did not occur in that place, but that the disease was contracted in Caterham.

Rightly looked at, I judge that the almost complete, if not the complete, exemption of Warlingham, is a very considerable confirmation of the view I have expressed with regard to the origin of this epidemic.

In the Table appended to this Report I have shown the daily distribution of the cases of enteric fever in all the places supplied with the Caterham Company's water. The total number of cases up to the end of February is 352. The total number of deaths is 21. After that date only a few scattered cases occurred.

Throughout, the disease was typical enteric fever, the patients exhibiting the characteristic rose spots and diarrhœa, and some of them suffering from severe pulmonary and intestinal complications, the latter including perforation of the bowels, which in four cases was the immediate cause of death. Notwithstanding this, however, by far the majority of the cases were of an exceptionally mild character, and although, when subjected to examination, they were ascertained to be cases of genuine enteric fever, it is certain that, had it not been for the prevailing epidemic, no medical advice would in many cases have been sought, and some of the patients would not even have taken to their beds. By far the majority of those attacked were children; and amongst adults, women were more frequently attacked than men. This distribution of the disease is by no means uncommon in epidemics in which water is the vehicle by which the infection is conveyed, for children drink much more water than adults, and amongst the latter, men as a rule drink less than women.

Remedial Measures. –

Immediately on ascertaining the circumstances under which it was evident that the water supplied by the Caterham Waterworks Company had become polluted, I took steps which led the Sanitary Authorities of the various districts concerned to issue notices informing the public that there were grounds for believing that the water in question had become accidentally contaminated, and cautioning them as far as possible, until further notice, not to use the water, or if no other supply could be procured, to have it thoroughly boiled before use. This notice was circulated on February 10th. I also advised the Waterworks Company to adopt the following measures:—1st, to pump their wells dry, if possible; 2nd, to scrape the walls of the wells and adits, afterwards scouring them thoroughly with a strong disinfecting fluid; 3rd, thoroughly to cleanse the reservoirs and wash them with disinfectants;

* The mean temperature on the grass, as registered by Dr. Adam at the Caterham Asylum from January 8th to January 31st inclusive, was 21·6° Fahr., it having varied from 12·0° Fahr. on the 11th to 31·5° Fahr. on the 15th; and the experience both at the Caterham Asylum and at the Guards Barracks is to the effect that during the late severe weather in January last the consumption of water was reduced nearly one half.

and 4th, to saturate with a similar fluid all the soil in the vicinity of their works where the chalk from the adits had been deposited.

Owing to the force which the springs supplying the wells exhibited at this time of the year, the Company, after long continued efforts in this direction, found it impossible to pump their wells dry. They, however, pumped to waste an amount of water considerably exceeding one hundred times the total contents of the wells and adits, and having materially lowered the water level, the walls of the wells and adits were thoroughly cleansed and scoured with a disinfecting fluid; large quantities of disinfectants being also thrown into the wells themselves. The reservoirs were thoroughly cleansed and disinfected, and the soil in the vicinity of the wells and reservoirs was again and again saturated with disinfecting fluid.* Measures were also taken so to flush the mains as to thoroughly clear them of all deposit. In short, the Company acted with considerable energy and promptitude in adopting all measures which were found possible to do away with the results of the accidental contamination to which their water had been subjected; and in view of the manner in which these measures had been carried out, I on March 5th communicated to the several Sanitary Authorities my opinion that, although in the absence of any test capable of showing the safety of the water it was difficult to speak on the point with authority, yet I thought the time had arrived when it might again be distributed by the Company without danger to the public health. I am glad now to add that nothing has since occurred which leads meto believe that the expression of this opinion was premature.

In all the sanitary districts concerned, the officers of the Sanitary Authorities visited every house where enteric fever was known to exist; disinfectants, with advice as to their proper use, were distributed, and besides other precautionary measures, immediate steps were in many cases taken to sever any direct connexions existing between the houses and either sewers or cesspools. To these steps, and as regards Red Hill to the efficiency of the system of sewerage, must probably be attributed the few secondary cases which arose. At Red Hill considerable energy was displayed in temporarily providing the public with another water-supply, water carts being used to convey it from house to house. In some parts of the district an alternative supply was not procurable. But everywhere people having private wells opened them freely to the general public. Some old wells, the contents of which were, to say the least, of a questionable character, and the use of which had very properly been abandoned for some time past, were re-opened, and the authorities should now endeavour to have them again closed.

During the course of the lengthened inquiry on which this Report is founded, I received the most ready co-operation from all whose assistance I found it necessary to seek. Throughout the district all the medical men regularly supplied either Mr. Jacob or myself with lists of their cases; the Chairman and Secretary of the Caterham Water Company placed all the information they possessed and the personal assistance of their staff at my disposal, and from the officers of the Sanitary Authorities I received similar help. To Mr. Jacob my thanks are especially due. The detailed inquiry, in so far as it related to the districts under his medical surveillance, was mainly conducted by him. The advice tendered to the Sanitary Authorities in those districts was the joint advice of us both, and the investigation was in several respects materially facilitated by the valuable suggestions which he made.

* Having regard to the stratum in which the wells and adits were constructed, chloride of lime was the disinfectant recommended for these several purposes Permanganate of potash was in addition also mixed with the water itself.

App. A. No. 8.

On Enteric Fever at Red Hill and Caterham, by Dr. Thorne Thorne.

TABLE showing DATES of ATTACKS from ENTERIC FEVER in undermentioned places, supplied with the CATERHAM WATER.

Date.	At Caterham.	At Red Hill.	At Earlswood Asylum.	At Godstone.	At Bletchingly.	At Nutfield.	At Warlingham.	Total.
Number of houses supplied with the Company's water -	419	924	1	39	28	34	156	—
1879.								
January 19 - -	1	—	—	—	—	—	—	1
„ 20 - -	1	2	—	—	—	—	—	3
„ 21 - -	—	3	—	—	—	—	—	3
„ 22 - -	—	5	—	—	—	—	—	5
„ 23 - -	2	12	—	—	—	—	—	14
„ 24 - -	3	6	—	—	—	—	—	9
„ 25 - -	3	16	—	—	—	2	—	21
„ 26 - -	8	7	—	—	—	1	—	16
„ 27 - -	3	13	—	—	—	—	—	16
„ 28 - -	3	11	1	—	—	1	—	16
„ 29 - -	4	6	—	—	—	2	—	12
„ 30 - -	1	2	3	—	—	—	—	6
„ 31 - -	2	17	3	—	—	1	—	23
February 1 - -	10	23	—	—	—	—	—	33
„ 2 - -	6	9	1	—	2	—	—	18
„ 3 - -	4	13	1	—	—	1	—	19
„ 4 - -	6	7	2	—	—	—	1	16
„ 5 - -	3	4	3	—	—	—	—	10
„ 6 - -	4	5	—	—	—	—	—	9
„ 7 - -	2	9	1	—	—	—	—	12
„ 8 - -	—	5	5	—	—	—	—	10
„ 9 - -	2	1	5	—	—	—	—	8
„ 10 - -	2	4	6	—	—	—	—	12
„ 11 - -	1	7	1	—	—	—	—	9
„ 12 - -	1	3	1	—	—	—	—	5
„ 13 - -	2	2	—	—	—	—	—	18
„ 14 - -		4	—	—	—	—	—	
„ 15 - -		5	—	—	—	—	—	
„ 16 - -		2	—	—	—	—	—	
„ 17 - -		3	—	—	—	—	—	
„ 18 - -	1	1	1	—	—	—	—	3
„ 19 - -	—	1	—	—	—	—	—	1
„ 20 - -	2	3	1	—	—	—	—	6
„ 21 - -	1	—	1	—	—	—	—	2
„ 22 - -	1	—	—	—	—	—	—	1
„ 23 - -	1	—	—	—	—	—	—	1
„ 24 - -	—	—	—	—	—	—	—	—
„ 25 - -	—	—	—	—	—	—	—	—
„ 26 - -	—	—	—	—	—	—	—	—
„ 27 - -	—	—	—	—	—	—	—	—
„ 28 - -	1	—	—	—	—	—	—	1
Date of attacks unknown - -	—	13	—	—	—	—	—	13
Totals - -	81	224	36	—	2	8	1	352

No. 9.

On an Outbreak of Scarlatina at Fallowfield, near Manchester, by Dr. Hubert Airy.

The following report relates to a sudden outbreak of scarlatina in the village of Fallowfield, three miles south of Manchester, of which the Board received information from Professor A. Gamgee, M.D., of the Owens College, Manchester, on August 12, 1879. The inquiry was taken in hand on August 16.

The main outline of the facts of the epidemic is as follows:—In the last week of July there were, among adults resident in Fallowfield, two or three attacks of illness (premonitory, so to speak, of the outbreak which followed) marked especially by vomiting and diarrhœa, and in one case by sore throat, but not developing the characteristics of scarlatina. On July 31 there occurred, in a child in one of the same families, a decided case of scarlet fever, which, however, could not be suspected of giving rise to any of the subsequent cases. On Sunday, August 3, two children of one family, living in Carill Drive (see map), and four children of another family living in Wilbraham (or Prince's) Road, were taken ill with symptoms which soon declared themselves to be those of scarlet fever, accompanied with vomiting and diarrhœa. In the course of Sunday night, August 3, and Monday, August 4, 18 persons in 11 other families, belonging to, or having relation with, though some of them absent from, Fallowfield, sickened in the same way. Several of them had felt their throats sore on the Sunday evening. On Tuesday, August 5, two other cases occurred in two families previously attacked. On Wednesday, August 6, another family had a slight case of sore throat, which might have been passed over had not two other members of the same family subsequently sickened with scarlet fever, apparently caught from the first by direct contagion, on August 9 and 14. There was also a doubtful case on August 7, and another on August 13.

Thus, including nine cases of somewhat doubtful or undeveloped character, such as are not unfrequently met with in connexion with epidemics of scarlet fever, there were in all 35 persons in 18 families attacked within a period of one month; and of these 35, no fewer than 24 were attacked within a space of 36 hours—between Sunday morning August 3 and Monday evening August 4.

A list of the cases is given at the end of this report, with a few notes of the attendant circumstances.

The symptoms which accompanied the onset of the disease were in many cases so remarkable as to lead the medical men who attended them to believe that there must be something exceptional in the cause or mode of infection. Not only was there frequent vomiting, but also severe and continued diarrhœa; indeed, in some of the persons attacked diarrhœa was the most prominent and almost the only symptom; and in one of the earliest cases (No. 4 in the list), which occurred in an adult and did not show full febrile development, it was remarked that the symptoms were more those of irritant poisoning than of scarlet fever. There was, however, no doubt at all as to the real nature of the disease in other cases which, with similar symptoms of irritation of the alimentary canal, presented also all the characteristics of scarlet fever. Among the medical men who were in attendance on these cases a decided opinion prevailed that in all probability the morbific matter had entered the system through the alimentary canal. The outburst of 24 cases of scarlet fever within 36 hours pointed to some one common source and

App. A. No. 9.

On Scarlatina at Fallowfield, by Dr. Airy.

agency of infection. At once suspicion fell upon the milk; and on inquiry it was found that every family that was attacked had received its supply of milk from one and the same dairy, while neighbouring families, taking milk from other dairies, had entirely escaped.

It was further found that one of the milkers employed at this dairy lodged at a house where a boy lay in the peeling stage of scarlet fever; and it was concluded that he had in some way conveyed the contagium of the disease to the milk.

Keeping these conclusions regarding milk (to which I shall afterwards return) as much as possible in the background, my inquiries were directed in the first place to the examination of other possible or conceivable modes of fever dissemination. To assist in the discussion of the several points to be considered, I will here refer to a sketch-map of the village of Fallowfield, which is appended to this report. The village is situated on low clayey land about half way between the rivers Medlock and Mersey. The centre of the village is occupied by a small but dense mass of dwellings of the poorer class, arranged in short streets, behind a row of shops which front the main road. Around this nucleus, in almost every direction, villa residences have been built, which are tenanted mostly by gentlemen having daily business in Manchester.

With only three exceptions, the families attacked with scarlet fever belonged to the better class, residing for the most part in detached villas, separated by large gardens from their neighbours. The houses in which scarlet fever appeared are distinguished by a red mark in the map. It will be seen that they are widely distributed in the village, about the northern part more than the southern, and especially about Wilbraham (or Prince's) Road on the west side of the main road, and Oak Drive, Carill Drive, and Ladybarn Road on the east. In these roads inquiry was made, not only at the houses where fever had been present, but also at those which remained free.

It was satisfactorily ascertained that the persons who were attacked with scarlet fever had not assembled together at any neighbourly gathering, garden party, or public entertainment within the widest possible limits of incubation of the disease. Indeed there had been no general assemblage of any such kind since Whitsuntide, the first week in June. Between the families K and M in Aucklands, communication was frequent, and between some of the families in Oak Drive, occasional. With these exceptions, intercourse between any two of the various families concerned had been exceedingly rare, if there had been any at all.

If all the persons attacked had been found to have attended the same place of worship on Sunday, July 27th, there might have been ground for suspicion that the infection had been contracted there from some previously infected member of the congregation. But there had been no such community of attendance.

If these 18 families had employed the same laundress, it might have been suspected that scarlet fever had been spread with the clothes from the wash. But it appeared that only two families out of the 18 had dealings with the same laundry, and in several of them the washing (especially of the children's clothes) was done at home.

There was no scarlet fever in the house of the postman who distributed letters in Fallowfield; nor in that of the newsagent who supplied most of the residents with daily newspapers.

There is no community of drainage between different parts of the district.

Water is supplied to the district not from a local source but from the Manchester Waterworks.

The prevailing wind for the last week in July and the first few days in August had been south-west, and in that direction there are several offensive ditches carrying sewage, but this influence, though unwholesome, is not known to be capable of causing scarlet fever.

But one and all of the above-suggested conceivable modes of conveyance of the fever disappear before the simple question, Why did the fever attack only customers of one particular dairy, when their neighbours, who were customers of other dairies, escaped? Exceptional incidence on families served from one dairy is, I need hardly say, not sufficient in itself to prove that the incident disease is spread from that dairy. If every family in a certain locality took milk from one dairy, it might easily happen that scarlet fever, due to some other local cause, might break out in that locality among families all taking the same milk; yet in that case the milk would not be the cause of the outbreak. But here, at Fallowfield, there are several different dairies, all having customers in the same localities. For instance, in Wilbraham Road, the suspected dairy (which I will call Δ) had the custom of five families; while 22 other families in the same road were served from other dairies. In Oak Drive six families were served from Δ, and five from other dairies. In Carill Drive, two families from Δ, and two from other dairies. In Ladybarn Road three from Δ, and six from others. But scarlatina attacked only families served from Δ. It is quite impossible that any local cause unconnected with the milk supply could have operated with such unerring selection upon the customers of one dairy, when so many who were supplied from other dairies were in all other respects exposed to the same local conditions. The conclusion is irresistible that the contagium of scarlet fever was spread *with* the milk from the dairy Δ;—we must not say *in* the milk until we have described the mode of milk distribution.

The families supplied from Δ were between 60 and 70, scattered over the whole village. With few exceptions they were families of the better class. Many of them were away from home at the time of the outbreak; others were on the point of leaving, and the date of departure in some cases will prove to be of critical value in determining the probable date of infection. On the annexed map the houses supplied from the dairy Δ are marked blue: those in which scarlet fever appeared are distinguished by a red mark. Those which are left blank were served from other dairies. It may be noticed that the whole village admits of division into two nearly equal groups, one to the north and the other to the south of Ladybarn Road; and it will be seen at once that the number of infected families in the southern group was only two, while in the northern group it was sixteen, including doubtful cases. I shall be able to show that the milk supply to these two groups was not exactly the same.

The position of the dairy farm Δ is shown on the annexed map. The sign Δ is placed against the shippen in which the cows were milked. I may say at once that there was not the slightest reason for suspecting any wilful dilution of the milk or want of cleanliness in the vessels. The business was perfectly honest, and there was general testimony to the richness of the milk. The cans were washed out with water laid on to the dairy from the Manchester waterworks. In this respect this dairy was superior to some others in the district. The cows were about 20 in number, and were, as far as was known, free from disease. The milkers were three in number; (1) the daughter of the farmer's widow who kept the dairy; (2) a young man who lodged in the farm house; (3) an old man who lodged at the house of his married son in Oak Street, in the crowded block in the centre of the village. Most of

the milking was done by Nos. 1 and 2. There was no definite order of milking; each milker took one cow or another as it might happen, till all were milked. The produce of each cow was poured from the milking-can through a straining cloth into the gathering-can, special care being taken as regards the cleanness of the cloth, and from it when full into one of two delivery-cans which were taken round by cart. Thus the first half of the milking was poured into one delivery-can, and the second half into the other. From the morning milking, two carts, each taking one delivery-can, were despatched on two separate rounds, one going to the right (northward), the other to the left (southward) from the bottom of the Ladybarn Road, one driven by milker No. 2, and the other by a nephew of milker No. 1, who himself was one of the persons attacked with scarlet fever on August 4th. From the afternoon milking, at 3 p.m., only one cart was despatched, taking two delivery-cans for the whole round. At the bottom of Ladybarn Road an additional quantity of milk was poured into one or other of the two Δ cans from a can (δ) belonging to a small dairy at Cheadle Hulme, six miles away. It could not be said whether the northern or the southern can was most likely to receive this addition; but it must not be overlooked that this addition would almost necessarily make some difference in quality according as the δ milk might be more or less pure than that from Δ) between the northern and southern supplies. After the can δ had been emptied, it was left standing on a flagstone by the roadside, and was picked up and taken back to Cheadle Hulme in time for the next milking there.

While the bulk of the customers were served by cart, those who resided near to the dairy Δ, in Aucklands (Ladybarn Road) and Carill Drive, were served by hand, the old man (milker No. 3) carrying round their daily supply morning and evening. Thus there was a distinction, as regards the *personnel* of the milk service, between these near customers and the rest; and also (in the morning) between the north and south groups that were served by the two carts. Now if the fever had been spread by direct personal contagion from one of the milk servers to the customers whom he served, we should have expected to find that spread confined to one or another of the three groups above mentioned. But none of these groups were exempt from the scarlet fever, and we may therefore conclude that the spread of the fever was not due to personal contagion. It seems, however, almost superfluous to insist on this point, for it could not possibly happen that so many children of so many families should come in contact with the person, whoever he was, that carried the milk.

We are driven then to the conclusion that the scarlet fever infection was conveyed *in* the milk supplied from the dairy Δ.

Though none of the three groups above mentioned were exempt from scarlet fever, yet it deserves careful notice that they were not all equally affected. The families that were personally supplied by milker No. 3 were 11 in number, including the farm Δ itself, and five of them were attacked with scarlet fever. Of the remaining six, three contained only servants, the families having left on or before July 20th, and in two the families consisted only of adults. In this group, out of six families that were probably susceptible of infection, five were attacked.

In the remainder of the north group, there were about 25 families supplied by the north cart in the morning. Of these, 10 were visited with scarlet fever. Of the remaining 15, four families had left, on July 9th, July 18th, July 19th, and August 1st, leaving only servants in the house, who took but little milk, and were generally supplied with it in the evening: one family had discontinued taking the milk from Δ on

July 29th ; at least six families consisted only of adults. In this group, out of 14 susceptible families, 10 were attacked.

The south group numbered about 30 families, of which only two were certainly infected. Of the remainder I am not able to give a precise account; but many had left, and several consisted only of adults.

This disparity will need to be taken into account in any attempt at explaining the origin of the outbreak.

As to the probable date of inception of the fever poison, we have evidence of singular importance in a remarkable case (No. 11) which has only recently come to light, a month after the date of the outbreak. I owe the particulars of it to Professor Gamgee, who received them from the physician (Dr. Thorburn, of Manchester), who attended the case. I give the account mainly in Dr. Thorburn's own words: On August 4th, a child, E. B., residing more than two miles from Fallowfield, was attacked with the scarlatinal rash and fever. There were smart premonitory symptoms on the evening of August 3rd. She had returned along with the rest of the family from an out-of-the-way country residence where scarlatina was untraceable, on August 1st. On Saturday August 2nd, she with four others of the children was sent to the farm Δ, whence the family intended to get their regular supply, and had there some milk to drink. She is more fond of milk than the others, and so it is probable that she had the largest quantity. This history, Dr. Thorburn remarks, taken in connexion with the Fallowfield outbreak, "seems to establish an almost certainty " that the child was infected by one drink of milk, that her brothers " and sisters who tasted more sparingly at the time were unaffected, " though one of them was very feverish for a few hours, and that the " period of incubation can have been little over thirty-six hours. " The last point is the most important. If any evidence could be " obtained of a similar character about other of the Fallowfield cases " it would tend to show that infection through the gastro-intestinal canal " implies a very short incubation."

Confirmatory evidence of the kind desired is not wholly wanting. A nearly parallel case is found in the family K. Two boys (Nos. 21 and 22), aged 14 and 16, returned from school at Wolverhampton, where there had been no recent scarlet fever, on Friday August 1st. On Saturday and Sunday, August 2nd and 3rd, they were at Fallowfield, and drank milk from the dairy Δ. On Monday August 4th, they left for Scotland, and both suffered from diarrhœa that night. On Tuesday the eldest boy had a rash. The fever was not fully developed in these cases, but they were very like some others in the same outbreak, and probably depended on the same cause.

Another case in which suspicion rested on one particular draught of milk occurred in the family C, where the lady of the house (No. 4) suffered from violent vomiting and diarrhœa in the night of August 2nd, having that evening taken some biscuit and a draught of milk for supper. The attack was so sudden and violent as to simulate irritant poisoning, and the biscuit at first came under suspicion ; the milk was overlooked until the general outbreak of the next few days became known.

Moreover, there was an instance of a family of three children, aged 4 months, 16 months, and 4½ years, living in Wilbraham Road, and drinking milk from Δ without any special precaution up to the morning of Friday August 1st (on which day they left home), yet escaping infection altogether.

A similar case occurred in Egerton Road, but this was in the southern group of customers, among whom the fever incidence was rare.

It should be mentioned, however, that another family of five children, of ages from 2 to 8, in Wilbraham Road, escaped altogether, though they did not leave home during the epidemic, and continued to drink milk from Δ. The milk was not boiled. I was unable in this case to discover any circumstance that would account for the exception.

While these cases go to show that infection in the main did not take place before Saturday August 2nd, there are three cases (Nos. 14, 15, and 26) in two families, H and N, which show that infection took place not later than about 2 p.m. on that day, for about that time these families left Fallowfield for distant parts of the country, and developed the symptoms of scarlet fever on Monday August 4th.

These indications conspire to make it probable that the principal date of infection was the morning of Saturday, August 2nd, the disease showing itself in 18 cases on Monday August 4th, after an incubation of some 48 hours, and in six cases on the Sunday, after, perhaps, only 24 or 36 hours' incubation.

We have now to inquire how it could have happened that the milk from the dairy Δ, and especially that portion of it which was delivered to the northern group of customers, should have become contaminated with the poison of scarlet fever.

There was no recognised disease among the cows; nor, if there had been, do we know anything as yet about the possibility of scarlatinal infection being derived from such a source. It would appear, then, that the contamination of the milk must have taken place either (1) in the act of, or after, milking at the dairy Δ, or (2) in the addition of the supplementary milk from Cheadle Hulme.

The latter alternative appeared at first sight full of promise, especially as it offered a probable explanation of the disparity of fever incidence on the north and south groups of customers; for it was sure to happen that this contingent from Cheadle Hulme should be added unequally to the north and south cans, and probably it would all be added to one of them. But on closer inquiry I gathered that the milk which was supplied to the nearest customers, and which was at least as infective as that which was supplied to the rest of the north group, did not contain any of the Cheadle Hulme milk, but was entirely the produce of the home dairy Δ. This fact is sufficient to clear the Cheadle Hulme milk from suspicion, and to bring the inquiry to a point on the dairy Δ.

I paid two visits to Cheadle Hulme, and examined into the state of things there. There was no scarlet fever in the house or among the milkers (who all live on the premises). The cans were rinsed with scalding water every evening. The water was got from a pump in the back yard, about 15 yards distant from the privy; it was clear and tasteless. The dairy business is on a small scale and simply conducted. All the milk (from five to eight dozen quarts a day) was sent to Fallowfield, except a few quarts which were supplied to three families close at hand. One of these three families, I was informed by their medical attendant, had two cases of scarlet fever, dating from the 14th and 17th of August. There had been a previous case in the same house 18 months before; but there was a good deal of scarlet fever in the neighbourhood, and the children might have caught it at the Sunday School.

Returning to the milk produced at the dairy Δ, I have already stated that early in the outbreak, investigation by two or three medical men in the neighbourhood, who were particularly interested in the matter, brought to light the fact that while at the farm Δ itself there was no scarlet fever prior to August 4th, and both milkers (Nos. 1 and 2) were free from any evident risk of contamination, the third milker, an old man named Hill, was lodging at a house where his grandchild was lying in the full height of desquamation after scarlet fever. The child lay in the back room on the ground-floor. The grandfather did not return to the

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house for his meals, but only to sleep. He always entered at the back door (the front entrance was practically unused), and it was not necessary for him to come in contact with the sick child, as the foot of the stair leading up to the room where he and his son slept together gave immediately upon the back door.

Having regard to the confined space, imperfect ventilation, low standard of cleanliness, and want of sanitary intelligence, it is difficult to believe that the old man Hill could come from that house without carrying about him the taint of scarlatinal infection. It is not at all impossible that he may have carried in his pocket a handkerchief, seldom changed, which had been in contact with, perhaps moistened by the mucus of, the sick child; and at any time his hand may have become accidentally charged with the scarlatinal contagium. What if some such accident happened on the morning of August 2nd, just before Hill took his share of the milking? Might not the taint have passed from his hand to the milk in the very act of milking?—or possibly from his clothing, as he grasped the milking can between his knees? There are evidently many possibilities of contamination under such conditions. The old man himself, I should say, vehemently denied that he had been near the child.

How then, on the theory that the milk was thus infected by one of the milkers at the dairy Δ, could it happen that there should be so great a disparity in the incidence of scarlet fever on the two groups of customers, north and south? It is plain that if the whole milking were all mixed together in one can, and the different customers were all served in the same way and under the same conditions, there would be no great inequality between the results in two groups, each containing so many individuals. But where there are two cans, there is at once the possibility of a difference (original or acquired) in their contents. And when one can is filled before the other, they not only receive the yield of different cows, but they must also receive in different degree the taint (if any) of the milkers' hands. It is indeed impossible that the contents of the two cans should be absolutely similar, though they may be equally wholesome or unwholesome; but in all probability they will be unequally so. In the case before us this inequality would in all probability be still further increased by the unequal addition of the Cheadle Hulme milk. It is, then, quite reasonable to suppose that, on the morning of Saturday, August 2nd, the two cans of milk which diverged at the bottom of Ladybarn Road were unequally infective, that the one which took a northerly direction was more strongly charged with the infection of scarlet fever than the one which went to the south. This supposition would account for the unequal incidence of the fever on the north and south groups. It must be confessed, however, that there is no positive evidence that the first part of the milking was delivered to the northern, and the last part to the southern group. By the time the inquiry had reached this stage, it became difficult to obtain information.

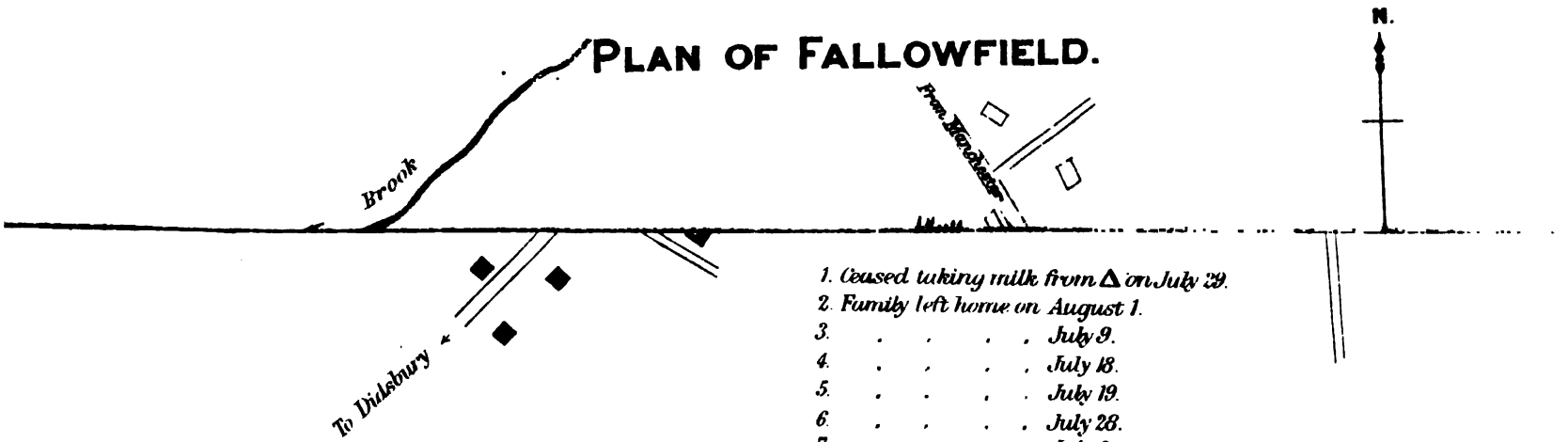
This, then, to sum up, is the theory which appears to offer the most probable explanation of all the facts of this remarkable outbreak. That the milkman Hill did in some way convey the infection of scarlet fever from his sick grandchild at home to the milk with which he was concerned at the dairy Δ; that while probably on one or two previous days the milk had been contaminated in a less degree, on one special occasion it was especially contaminated; that the contents of the milk-can from which the northern group of customers were supplied were on that occasion contaminated in a higher degree than the contents of the other can from which the southern group were served, and that thus there was more general spread of fever among the former than among the

latter; that this special contamination probably took place at the morning milking on Saturday, August 2nd; and that the period of incubation in the majority of cases was about 48 hours.

List of Cases of Scarlet Fever in Outbreak at Fallowfield.

No. of Case.	Place of Abode.	Family.	Age.	Date of Attack.	Nature of Attack and Notes.
	Oak Drive - -	A.	Adult	July 24, 28	Diarrhœa and vomiting.
2	Aucklands - -	B.	„	July 30	Sore throat. Vomiting on Aug. 3rd possibly from milk taken on Aug. 2nd.
3	Do. - -	„	9	„ 31	Vomiting, scarlet fever. This family B lived nearest to the farm Δ, and took large quantities of milk every day. A younger child, who was sent away on Aug. 2nd, escaped.
4	Wilbraham Road -	C.	Adult	„ 26, Aug. 2	Diarrhœa and vomiting on Aug. 2nd after milk and biscuit for supper.
5	Carill Drive - -	D.	5½	Aug. 3	Scarlet fever.
6	Do. - -	„	8½	„ 3	
7	Wilbraham Road -	E.	7	„ 3	
8	Do. - -	„	8	„ 3	Diarrhœa and vomiting. Severe scarlet fever.
9	Do. - -	„	12	„ 3	
10	Do. - -	„	13	„ 3	
11	St. Saviour's, Manchester.	F.	?	„ 4	Returned from the country, where no scarlet fever, Aug. 1st. Had a draught of milk at Δ on Aug. 2nd. Smart premonitory symptoms on evening of Aug. 3rd, rash and fever on Aug. 4th.
12	Oak Drive - -	G.	12	„ 4	Attacked with scarlet fever in the morning; had complained of sore throat the previous evening.
13	Do. - -	H.	25	„ 4	Attacked in the morning with sore throat. Scarlet fever.
14	Do. - -	„	22	„ 4	Sore throat in night Aug. 3rd–4th Scarlet fever.
15	Do. - -	„	15	„ 4	The two last left home on Aug. 2nd at 2 p.m., and were taken ill at Pwllycrochan in North Wales.
16	Wilbraham Road -	C.	3	„ 4	Diarrhœa and vomiting. Severe scarlet fever. In this family an infant, for whom the milk was boiled, escaped.
17	Do. - -	I.	10	„ 4	Left home in the morning, taken ill in the evening at Coventry. Scarlet fever.
18	Do. - -	„	11	„ 4	The last had complained of sore throat before leaving.
19	- - - -	„	15	„ 4	Scarlet fever. Sore throat the previous evening.
20	Aucklands - -	K.	11	„ 4	Scarlet fever. Diarrhœa and vomiting about Aug. 6th.
21	Do. - -	„	14	„ 4	Returned from school at Wolverhampton, where no scarlet fever, Aug. 1st. Left home for Scotland Aug. 4th. Both had diarrhœa. The elder had a rash on Aug. 5th.
22	Do. - -	„	16	„ 4	This family took large quantities of milk from Δ.
23	Sherwood Inn -	L.	7	„ 4	Sore throat; feverish.
24	Aucklands - -	M.	3	„ 4	Scarlet fever.
25	Do. - -	„	4	„ 4	Scarlet fever.
26	Egerton Lodge -	N.	6	„ 4	Family left home on Aug. 2nd for Pitlochry. Child attacked there on Aug. 4th with scarlet fever. Four other children escaped, one had had scarlet fever before.
27	Large Oak Farm (Δ) -	O.	12?	„ 4	Scarlet fever. A sister and a cousin escaped.

PLAN OF FALLOWFIELD.



1. Ceased taking milk from Δ on July 29.
2. Family left home on August 1.
3. . . . . July 9.
4. . . . . July 18.
5. . . . . July 19.
6. . . . . July 28.
7. . . . . July 2.
8. . . . . July 20.
9. . . . . August 1.

■ Families supplied with milk from the Dairy Δ

◧ Dº but no children, or only a servant or servants left in the house. (This distinction is not made in the southern part of the district.)

□ Dº supplied from other Dairies. (In the extreme south of the district these are not shown.)

✕ Against houses in which Scarlet Fever (or illness apparently due to the same cause) appeared. The letters refer to the families so de-noted in the List of cases.

● Against Nº 4, Oak Street, where milkman lodged.

To accompany Dr Airy's Report on Fallowfield.

DANCER FIELD, LITH. 22 BEDFORD ST COVENT GARDEN 695

No. of Case.	Place of Abode.	Family.	Age.	Date of Attack.	Nature of Attack and Notes.
28	Portland Grove -	P.	6	„ 4	Scarlet fever.
29	Aucklands - -	M.	5	„ 5	Scarlet fever.
30	Oak Drive - -	H.	Adult	„ 5	Scarlet fever.
31	Egerton Road -	Q.	8½	„ 6	Sore throat.
32	Main Road - -	R.	5	„ 7	Sore throat.
33	Egerton Road -	Q.	12½	„ 9	Scarlet fever, ? caught from her sister, No. 31.
34	Oak Drive - -	S.	3½	„ 13	Feverish, ? scarlet fever. Had been in company with No. 13 on Aug. 2nd.
35	Egerton Road - -	Q.	17	„ 14	Scarlet fever, ? caught from No. 33.

App. B. No. 1.

On the Lymphatic System of the Skin and Mucous Membranes, by Dr. Klein.

APPENDIX B.

No. 1.

On the Lymphatic System of the Skin and Mucous Membranes, by E. Klein, M.D., F.R.S.

In No. III. (New Series) of the Reports of the Medical Officer of the Privy Council and Local Government Board, I gave an extensive summary of an anatomical investigation of the lymphatic system of the serous membranes and the lung, carried on during 1871 and 1872, with special reference to the process of chronic infective inflammation and tuberculosis. These investigations have been published in an extended form, with the sanction of the then Medical Officer, and by the aid of the Royal Society, as "The Anatomy of the Lymphatic System, Part I., The Serous Membranes, 1873; Part II., The Lung, 1875."

With the permission of the late Dr. Seaton and the present Medical Officer, I have been able to resume these investigations and to extend them, during the past two years, to the lymphatic system of the skin and mucous membranes, and on these I propose to report in the following pages.

I.—The Lymphatics of the Skin of Man and Mammals.

1. In accordance with the observations of Teichmann, published in his classical work: Das Saugadersystem vom anatomischen Standpunkte, Leipzig, 1861, I. Neumann, in his recent investigations of the lymphatics of the skin (Zur Kenntniss der Lymphgefässe der Haut, &c., Vienna, 1873), also distinguishes the superficial from the deep-seated lymphatics, both forming a plexus, *i.e.*, the superficial and the deep plexus. The former is denser and its vessels finer than in the latter. G. and F. Hoggan (Proceedings of the Royal Society, No. 182, p. 289*) describe the lymphatics of the mammalian skin as consisting of a plexus of "subepidermic" and one of "subhypodermic" vessels; both are connected by the "dermic" vessels which form horizontal and vertical sets.

All these authors agree that the superficial vessels are capillaries, while the deep ones are the efferent vessels possessed of valves, and G. and F. Hoggan consider also the "dermic" vessels as possessed of valves.

Both Teichmann and Neumann noticed lymphatics in the papillæ of the corium; these are branches of the superficial plexus. According to the former the papillæ of the hand and foot contain each a lymphatic vessel terminating in a cœcal extremity, but always less superficially than the blood capillaries. Neumann saw in all well-formed papillæ a single lymphatic terminating either in a cœcal extremity or a loop.

G. and F. Hoggan deny the existence of these papillary lymphatics, but this is probably owing to their having studied only the skin of mammals, which generally do not possess well-formed papillæ. In well injected specimens of mammalian skin, however, indications of papillary lymphatics are undoubtedly present (*see* below).

2. The lymphatic vessels referred to here and in the following pages as the deep efferent vessels are distinct from, and much finer than the

* I have not been able to find where the full paper of these authors is printed, and I am therefore limited to the "Abstract" published in the Proceedings.

vessels which, being the collecting vessels of large cutaneous districts, run between the skin and the subjacent organs, as fasciæ, tendons, &c. These tubes are possessed of a comparatively thick wall, composed besides the lining endothelium, of an elastic intima, a circular muscular media, and a thin connective-tissue adventitia. In this respect they conspicuously differ from the lymphatics belonging to the skin, the wall of which is merely a single layer of endothelial plates, either more or less elongated with straight or sinuous outlines, as in the deep and larger, or sinuous in shape, as in the superficial smaller vessels.

The collecting vessels possess, as shown by Biesiadecki, their own system of blood-vessels, consisting of an arterial and venous branch, and a network of capillaries.

A. Dogiel quite recently (Archiv. f. Mikr. Anatom. vol. xvii. p. 335) demonstrated the existence of a network of blood capillaries surrounding the large efferent (collecting) vessels underneath the skin of the earlobe of the hind extremity of the rat, in the mesentery of the cat and the dog.

3. In studying the lymphatics of the skin, it is necessary, as will be shown by-and-bye, to consider them in their relation to the various organs of which the skin consists, for they, viz., the lymphatics, as well as the blood-vessels, as shown by Tomsa, stand in a special relation to those organs.

We shall, then, consider them as: A. The lymphatics of the connective-tissue matrix of the skin; B. those of the fat-tissue; C. those of the sweat-glands; and D. those of the hair-follicles, sebaceous glands and arrectores pili.

Such an attempt of distinction has also been made by Neumann, but as I shall show, what this observer described as the lymphatics of the fat-tissue, hair-follicles, sebaceous glands, and sweat glands, are merely the lymphatics of the surrounding connective-tissue, and do not in strictness bear any other relation to those organs than that of efferent vessels. The lymphatics that properly belong to those organs have not been known to Neumann or to his predecessors.

The assertions of G. and F. Hoggan as to the non-existence of lymphatics in the fat-tissue, hair follicles, sebaceous glands, arrectores pili and sweat glands have, like most negative results, only a relative value, inasmuch as they show, what, however, did not require any special proof, that whilst it is comparatively easy to demonstrate the lymphatics of the connective-tissue ground substance of the skin, those of the fat-tissue and the glandular structures require special methods and attention.

A.—The Lymphatics of the Connective-tissue Ground Substance.

4. These are the lymphatics that have been known to all observers, and have been specially studied through injections with various colouring matters by Teichmann, Neumann, and others. They have already been referred to. Teichmann as well as Neumann (and also G. and F. Hoggan) consider the above lymphatics as the ultimate vessels, and as not possessing any direct connexion with the surrounding connective-tissue. That they should have arrived at such a conclusion with reference to the connective-tissue need not surprise us, seeing that they were not able to demonstrate the ultimate vessels belonging to the fat-tissue or the glandular structures of the skin.

5. The methods used by Teichmann and Neumann seem to be altogether unfavourable for the demonstration of the ultimate lymphatics. This is the method followed by Neumann (l.c., p. 8): "The method "that I used was the modified method of Hyrtl-Teichmann; the "epidermis having been removed by maceration in a mixture of alcohol,

"acetic acid, and water, and also the rest of the tissue having become "macerated, an incision half to one line deep is made with a pointed "cataract needle, into which a fine tubule is fixed, and the injection "through a fine brass syringe is proceeded with. . . . ." As injection material Neumann used (l.c., p. 9) carmine-ammonia in conjunction with glycerine, and carbonate of lead rubbed down in a little glycerine. On p. 10 he informs us that to prevent mistaking the lymphatics for blood-vessels, he injected the latter with Berlin blue. But to our profound astonishment we find in the beautiful plates accompanying his memoir, all lymphatics represented as if injected with Berlin blue, although this pigment, according to his statement, is reserved solely for the injection of the blood-vessels. These latter are represented (in the plates) as if injected with carmine-ammonia, but according to his statement this is the material which he used for the injection of the lymphatics.

After this and after his mentioning (l.c., p. 9), that it is advisable to stretch the skin that is to be injected over the left finger during each injection, we cannot expect that Neumann should have been able to trace the ultimate rootlets of the lymphatics.

6. The method which I follow is the same in the case both of the skin and mucous membranes. It is this: I always use the material as fresh as possible; my most successful specimens were obtained from skin and mucous membranes prepared immediately after death. This applies equally to the structures of man and animals. To use material that had been previously macerated in alcohol and acetic acid, as in the case of Neumann's material, and to expect that the injection would reveal the finest rootlets, appears to me too sanguine.

Human skin is best; that of the dog and rabbit is also good. Of the former the scalp, the skin of the face, of the lips, of the palm of the hand and that of the sole of the foot are those parts that yielded the best preparations.

The method of preparation is that of injection by "puncture." This is always performed on the skin in situ, not on skin cut out previously and stretched over the finger (!) as is done by Neumann.

7. The injection material used is Brücke's Berlin blue in a solution of two to four per cent, or asphalt dissolved in benzole on the plan of A. Budge (Archiv. f. Mikr. Anat., Band XIV., p. 70). This latter is an excellent material for injecting the finest rootlets of the lymphatics, for it flows with great ease and rapidity and has no influence, hardening or otherwise, on the tissue. In these two respects it is greatly preferable to any other injection matter. Berlin blue or nitrate of silver both possess, especially the latter, a hardening influence, and cannot be expected therefore to flow as easily into the most distant and finest rootlets as the asphalt-benzole solution. The most successful injections of the finest rootlets in the connective-tissue, fat-tissue, hair-follicles, and glandular organs have been obtained by this last named reagent. I must however add that in some cases—even with the asphalt-benzole solution—only the lymphatics of the superficial and deeper plexuses became injected, the injection matter not having penetrated into the ultimate rootlets, although the former were widely distended.

The solution is made thus: a very concentrated solution of asphalt in benzole is prepared, and before using it is mixed with ½ to 1 of its volume of benzole. Asphalt in chloroform, as originally employed in C. Ludwig's laboratory, is not so advantageous, and still less asphalt in turpentine.

8. The injection is carried out in this manner: A fine steel canula, as fine as it can be got, filled with the injection material, is gradually

pushed in a very oblique direction, as horizontal as possible, into the *corium itself*. Injections into the deep subcutaneous tissue do not yield good results, at any rate even in the most successful injections one does not generally obtain much more than an injection of the deep large lymphatics.

An ordinary hypodermic syringe filled with the injection matter, and fitting into the canula, is then gently and gradually emptied into the canula. In a successful injection the skin becomes very gradually thicker and swells up, without becoming bulged out; the swelling spreads gradually and uniformly into the periphery. *The mouths of the hair-follicles become very conspicuous as depressions* owing to the parts between them gradually projecting beyond the general surface. And this is one of the best signs of a successful injection. Continuing the injection the skin becomes still more swollen, and the circumference of the injection also increases accordingly.

Owing to the opacity of the epidermis it is not possible even with a lens to see the lymphatics themselves, the skin presenting a uniform colour, brownish black if asphalt, blue if Berlin blue is used.

The intensity of the colour and the swelling up of the injected skin is always greater in the former case, *i.e.*, asphalt, than in the latter, *i.e.*, Berlin blue. But of course this depends on the amount of the injected material and on the ease with which it spreads in the skin, for if a relatively large quantity is injected which does not spread into a comparatively large area, the skin will be deeply coloured and become very thick, owing to the lymphatics being greatly distended by and filled with the injection matter.

In the description of the appearances of the lymphatics, to be given below, we shall refer only to those preparations in which the injection has been effected with great ease, the skin and mucous membranes having become readily injected, and slightly but distinctly thicker than in the uninjected state; the lymphatics are then found in a moderate state of distension.

In the case of the mucous membranes where the lymphatics are easily recognised in the injected organs with the unaided eye, the successful progress of the injection soon becomes apparent, for the vessels become suddenly filled over large areas.

9. The skin and mucous membranes after the injection are hardened in dilute spirit—three parts of methylated spirit, to one part of water; or in a mixture of two parts of $\frac{1}{6}$ p.c. watery solution of chromic acid and one part of methylated alcohol; or in $\frac{1}{6}$ to $\frac{1}{4}$ p.c. solution of chromic acid only. When sufficiently hard, sections are made, stained, and mounted in the usual manner.

There are, however, certain disadvantages connected with the use of the asphalt-benzole. In the first place it is not so easily managed as watery solutions, and this of course applies to all other similar materials dissolved in turpentine, chloroform, oil, &c.; moreover the cleaning of the syringes, canulas, tubes, and instruments requires a deal of time and attention.

In the second place the difficulties of mounting and preserving the sections of the injected skin are considerable. These can be of course mounted and preserved in glycerine without any difficulty, but the section lacks transparency and smoothness of appearance of the injected vessels. The sections cannot be preserved in Canada balsam or damar varnish, on account of the solubility of the asphalt in balsam, turpentine, and chloroform.

I have succeeded, however, in preserving specimens in a transparent state for several months in the following way. The sections, after staining,

are treated in the usual way for mounting in balsam or damar, viz., they are placed in absolute alcohol and then in oil of cloves, where they obtain the desired transparency. The section having been lifted out of the oil of cloves and the excess of the latter having been allowed to run off, is mounted in glycerine-jelly. For the first month or two the lymphatics retain the asphalt in its original (black) state, but this becoming partly dissolved the vessels appear then filled with a paler (yellowish-brown) material. After several months the material still more fades into a transparent yellow, but the vessels remain nevertheless distinct.

In isolated cases I have been able to preserve some of the asphalt injection, of course only faintly brown in colour, in the lymphatics of the skin of the new-born child, even in sections mounted in Canada balsam solution.

10. It is hardly necessary specially to add that in the examination of sections, both through the injected skin and mucous membranes, only such parts have been selected as are at some distance from the point of "puncture," so that artificial extravasations may be excluded.

In the description of the lymphatics of the connective-tissue ground substance of the skin we shall take first those of man.

11. In a vertical section through the skin, *e.g.*, of the hand or foot, scalp, or of the face, where the lymphatics appear well injected, most of these present themselves cut transversely or obliquely. This would of course indicate that most of them possess a more or less horizontal direction, *i.e.*, parallel with the surface. There are, however, connecting vessels to be seen in all layers which run for a shorter or longer distance a longitudinal course. *From the papillary body down to the layer of the adipose tissue the connective-tissue of the skin in a vertical section appears uniformly pervaded by these lymphatics*, except of course where it is interrupted by the presence of the hair-follicles, sebaceous glands, and sweat glands. The division into a superficial and deep stratum, as maintained by other observers, cannot be well maintained.

12. J. Neumann, in his memoir above-quoted, gives in Fig. 1, Plate I., an illustration of continuous plexuses of lymphatics in a *vertical* section through the skin of the finger of the new-born. Such appearances, viz. a continuous plexus of lymphatics in a *vertical* section, I have not met with in any preparation of the skin in either man or mammal, and I doubt whether Neumann's figure is drawn from an actual vertical section or is merely a diagrammatic representation only. The section requires to be very oblique, in order to show only approximately so many longitudinal vessels as are here drawn. No vessel is shown in this figure in transverse section, whereas most lymphatics from Neumann's own description follow a horizontal course.

13. As regards the size of the vessels seen in one vertical section, we find, as a general rule, that the majority of the vessels belonging to the deepest layer of the corium, that is the layer containing the hair-bulbs and sweat glands, are larger than those of the middle layer; those of the latter again smaller than those of the most superficial layer, *i.e.*, of the papillary body, bearing in mind that we are here speaking merely of their relative numerical proportion and of preparations of which the lymphatics have been distended to a medium degree.

14. The papillæ, especially where they are well developed, as in the fingers, scalp, &c., contain, as already described by Neumann, lymphatics either apparently terminating in pointed or cœcal extremities, or as a single or even double loop; and I have seen papillæ in the finger of a new-born child containing complex loops of lymphatics, *i.e., a special network of their own.* Sappey mentions a similar relation with regard to the lymphatics of the papillæ. In those parts

of the skin of the new-born that do not contain well-developed papillæ, the lymphatics are indicated by short prolongations of the most superficial lymphatics.

The best view of the lymphatics of the papillæ is obtained in a thick vertical section.

15. Making a series of horizontal sections from the papillary layer down into the adipose tissue we find *in each section a beautiful and more or less uniform plexus of lymphatics*, the vessels varying very much in size. The plexuses are of course interrupted by the presence of the hair-follicles and glands. Corresponding to the above-named appearances in the vertical section, viz., that most lymphatics run a course parallel to the surface, we find them in a horizontal section chiefly longitudinal. Only few vessels are seen cut transversely or obliquely, that is those running vertically or obliquely. In all strata we meet with vessels which appear to terminate *in the tissue*. Of these are at once to be omitted those which on focussing can be seen to be cut away obliquely, that is, are running to the next upper or lower stratum, but there always remain a good many vessels of which this cannot be said, and which really terminate in the tissue. They are generally running out into a narrow cleft or channel, or terminate with a cœcal extremity, as is the case in some papillæ (*see* below). As a general rule we find the majority of the vessels of the most superficial stratum larger than those of the middle layers, and these again smaller than those of the deeper sections. I cannot agree with Teichmann, Neumann, and others, that the lymphatics are arranged as a superficial and deep plexus, and that those of the former are finer and their plexus denser than that of the deeper. I maintain, on the contrary, that throughout the thickness of the skin there are horizontal plexuses of lymphatics, of course with oblique or even vertical connecting branches; that the vessels of the superficial layers are larger than those of the middle layer; and that there is no absolute difference in the density of the plexuses, for the vessels are tolerably densely arranged in all layers, but relatively less so in the deeper strata, owing to the interposition of the sweat glands and the hair bulbs.

16. As regards the shape and nature of these vessels, they appear more or less tubular, but many of them (and this refers to all layers, including the papillary stratum) are irregular, since they show isolated saccular dilatations, or appear through a succession of them, more or less varicose. In all layers very fine vessels are seen to join tubes many times larger, and one and the same vessel if followed for a distance may be seen to change its calibre. In these respects the lymphatics comport themselves like those of other organs. Following in a horizontal section under a higher power the outline of some vessels, especially of those that run out freely into the tissue, the outline will be found not quite smooth, but showing from place to place minute discontinuities and irregularities, which give the vessel a more or less crenate appearance. This has also been observed by Schenk, and its significance will be explained later on.

All lymphatics are lined with a single layer of elongated endothelial plates, and in this respect they do not differ from other lymphatics. Besides this endothelium there is no other tissue which can be called their own, except the trabeculæ of the ground substance, forming their outer support.

17. Teichmann, as well as Neumann, maintain that only the lymphatics of the deep subcutaneous plexus are possessed of valves.

G. and F. Hoggan ascribe such also to their "dermic" vessels, that

is, to those extending as horizontal and vertical sets of vessels through the whole thickness of the skin.

I agree in this respect with G. and F. Hoggan, inasmuch as I find that there are lymphatics with valves in all layers of the skin.

18. Continuing the horizontal sections below the subcutaneous tissue, *i.e.*, into the adipose tissue, we find that the connective-tissue septa between the lobules of the latter, which, in the new-born child, are very well marked and regularly arranged, contain *very numerous lymphatics*. These in well injected specimens appear as plexuses of densely placed vessels and clefts, situated between the lamellæ constituting those septa. They will be later on described as the interlobular lymphatics of the adipose tissue. The vessels are smaller than those of the subcutaneous tissue, and they pass into the plexus of large lymphatics situated underneath the adipose tissue.

19. A very interesting observation that I made in the skin of a child that had died during or very soon after birth was this. The skin was covered, as usual, with a thick layer of the secretion of the sebaceous glands, but in vertical and horizontal sections through the hardened skin lymphatic vessels in all different layers could be seen, *containing in their interior small and large clumps of what was undoubtedly the same sebum as that on the free surface.* In the large lymphatics of the depth this was especially very easily ascertained. In Fig. XII. will be found lymphatic trunks containing such clumps of sebum.

We shall presently have to return to this fact, and it will then become clear how this sebum found its way into the lymphatics, and of how great importance this is in the understanding of the absorption from the external surface by the lymphatics of the skin. At present we wish to point out that the mouths of the hair-follicles were greatly distended by, and filled with this sebum.

20. The lymphatics of the skin of the dog are similar in their arrangement to those of man, and for this reason we shall be able to be brief in their description, especially since we can refer the reader to Figures IV., V., VI., VII. of the Plates accompanying this report. These illustrations and their explanation (printed at the end of this paper) give an accurate and sufficiently clear idea of the distribution and nature of these lymphatics. It will be seen that the lymphatics even of the most superficial layer (Fig. V.) are large vessels, some of which present all appearances associated with valves in lymphatics, viz., saccular dilatations and corresponding constrictions. The vessels of this superficial layer are very densely placed, and therefore of short extension. Owing to the arrangement of the hairs in groups of four to six or more hair follicles of unequal thickness, the plexuses of lymphatics are not uniform, being limited to the connective-tissue separating or surrounding these groups.

The septa of the adipose tissue possess fewer interlobular lymphatics than in man.

21. In what connexion do the above lymphatics stand to the blood vessels? Both in the skin of man and mammals, but more especially in the former, I find in all strata of the corium, notably in the middle strata, some lymphatic vessels possessing a more or less intimate relation with the blood-vessels. This relation consists in the following: (*a*) we find a blood-vessel, either an arterial or venous branch, ascending to, or descending respectively from the surface of the corium, accompanied for a longer or shorter distance on one or both sides by one or two lymphatic vessels; or (*b*) the blood-vessel is crossed by one or two or even more small lymphatics in a very oblique direction, and the latter appear at this point as if belonging to the sheath of the former.

Around the larger arterial branches we find numerous lymphatics of varying size, which in some instances form a more or less complete sheath around the blood-vessel,— perivascular lymphatics.

22. In what relation do the lymphatics stand to the general connective-tissue matrix, or in other words, what is the nature of the lymphatic rootlets? Brücke, Ludwig, v. Recklinghausen were the first who maintained the origin of the lymphatics in the clefts of the connective tissue.

These clefts, situated between the bundles or groups of bundles of the connective-tissue, have been injected in many organs naturally and artificially, and have been shown to be connected with the lymphatics. Without regarding it necessary to enter into a detailed historical review of this question, I will only mention the researches of v. Recklinghausen, Key and Retzius, of Sikorsky, myself, Arnold, Sachs, Küttner, and others, by which it has been conclusively established that the interfascicular spaces, *i.e.*, the clefts and channels between the bundles and groups of bundles of connective-tissue fibrils, containing in a semi-fluid interstitial albuminous substance the flattened connective tissue cells, are the paths through which the normal current of plasma, irrigating the connective tissue, proceeds, and that, through this current, fluid as well as formed material is carried into the lymphatics. How is this, then, effected? How are the interfascicular clefts and spaces connected with the lymphatics?

As I have pointed out in my Anatomy of the Lymphatic System in agreement with v. Recklinghausen, Sikorsky, and others, the lymphatic capillaries connected on the one hand with the plexus of lymphatics, and originating on the other hand in the tissue, open everywhere freely into the interfascicular spaces, their lumen communicating freely with the latter.* This accounts for the endothelial wall being here perforated by numerous holes or stomata. The identical condition is met with in the intertubular lymphatics of the testis (v. Mihalkovitch) and in the connective tissue of the membranes of the brain and spinal cord (Key and Retzius), where lymphatics are lined with perforated endothelium, the holes leading into the spaces between the connective-tissue bundles. But there is also another indirect mode of connexion of the lymphatic with the interfascicular spaces, and that is the mode which no doubt is the more common one, which occurs in the lymphatics in all strata. It is this: the albuminous cement-substance between the endothelial cells forming the wall of the lymphatic is continuous with the similar substance acting as cement between the connective-tissue bundles. This substance being soft and semi-fluid does not present any obstacle to the passage of either fluid or formed matter from outside, *i.e.*, from the interfascicular spaces into the lymphatic vessel.

This mode of connexion is the one by pseudostomata in contradistinction to the former or direct one by real stomata.

This relation plays an important part in the absorption of chyle globules by the chyle-vessels of the intestinal villi (Watney), and the same relation also exists between the lymphatics and the epithelium of the surface of the mucous membranes and glands (Sikorsky, Klein, Arnold, Thoma, Küttner, and others), and the endothelium of the surface of the serous membranes, as I have shown in my Anatomy of the Lymphatics.

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* The above-named crenate and irregular outline of some of the lymphatics is due to this fact, viz. that their endothelium is perforated, and their lumen opening into the spaces of the surrounding connective-tissue.

The assertions of Neumann and of G. and F. Hoggan that the lymphatic vessels are closed tubes not in communication with the surrounding connective-tissue refer only to their respective preparations: they are not applicable to what I find in my preparations in which the lymphatics are successfully injected with an asphalt-benzole solution. Both in the skin of the dog, pig, rabbit, and especially in that of man, I find clear proofs of the free opening of lymphatic capillaries into the interfascicular spaces (*see* fig. XV., XV.A). But also in some instances of Berlin blue injections I have traced most decidedly the Berlin blue from the interfascicular spaces into lymphatic tubes, as is represented in Fig. IX.

Very interesting and decisive proofs of this connection I obtained from preparations in the case of the new-born child above mentioned, whose lymphatic trunks contained clumps of sebum. Here I found in many parts of the skin *smaller and larger drops and clumps of sebum contained in the interfascicular spaces*, being either unstained or stained brown with the asphalt-benzole. In some places the interfascicular spaces were filled with the sebum particles, and we had then a *good natural injection of the interfascicular spaces. They could be easily traced into the lymphatic vessels.*

B.—*The Lymphatics of the Adipose Tissue.*

23. The adipose tissue is very richly supplied with lymphatics, and the investigation of these forms one of the important points of this inquiry. Teichmann knew of no lymphatics in the fat-tissue, and G. and F. Hoggan also deny the existence of such. Neumann mentions in his monograph above quoted (l.c., p. 25, cap. III., fig. 5) that the lymphatics form wide meshes which surround the individual lobules of the fat-tissue, but he did not succeed in tracing lymphatics between the fat cells, *i.e.*, into the interior of the lobules. The vessels seen and described by Neumann are, as I have already pointed out, merely lymphatics belonging to the connective-tissue septa between the lobules of the fat-tissue, and for this reason they may be described as the *interlobular lymphatics*. Comparing Neumann's drawings with my own, I am led to conclude that he saw very much fewer interlobular lymphatics than I have done. I have also drawn attention to the difference in numbers of these interlobular lymphatics in the fat-tissue of the human skin and in that of mammals, they being greatly more numerous in the former than in the latter.

The interlobular lymphatics take up everywhere *fine lymphatic sinuses and clefts which are traceable between each two fat-cells.* In specimens successfully injected with Berlin blue, and especially in those with asphalt-benzole, these *intralobular* or *intercellular* lymphatics are shown with great distinctness. I may particularly draw attention to Fig. IV. 3, and Fig. VII., illustrating the ultimate lymphatics in the fat-tissue of the skin of the dog, and Figs. XIII. and XIV., showing the same in man.*

In Fig. XIV. the injection matter (Berlin blue) might be said to have accidentally (by extravasation) passed in between the fat-cells, but this cannot be maintained for Fig. VII. and Fig. XIII. Here the connexion

* The asphalt-benzole, in a successful injection of the fat tissue, penetrates generally a good distance into the lobule, but the different lobules vary greatly in this respect, for I have seen in the same section, both of human and dog's skin, lobules, the greater part of which was injected, besides others in which only the peripheral portions showed the lymphatic injection.

between the intercellular lymphatics and the lymphatic vessels of the septa between the fat-lobules is as clear as one can desire.

24. Several important questions present themselves in connexion with these intralobular or intercellular lymphatics: (*a*) what is their shape and minute arrangement? (*b*) what is the nature of their wall? and (*c*) what is the relation of the latter to the fat-cells themselves?

As regards their minute arrangement, it is that of a dense network whose meshes each contain one fat-cell. It is more difficult to ascertain their precise shape. At first sight these intercellular lymphatics appear as thin narrow canals, but on closer inspection it will be found that many are in reality sinus-like, surrounding the fat-cells for a larger or smaller section of their circumference, and therefore possessing the shape of a portion of a spheroid. Some of them, however, are tubular or approximately so, their optical section being either circular or more or less oval.

In specimens of fat-tissue successfully injected with asphalt-benzole, it will be found that the injection material indicating the intercellular lymphatics is distinctly contained in a well-defined space, there being a delicate but distinct membrane limiting it. But it is exceedingly difficult to ascertain what this membrane is, viz., whether it is an endothelial membrane representing the proper wall of the lymphatic, or whether it is merely the membranous connective-tissue between the fat-cells. I am inclined to think the latter is the correct view. In very thin and well-stained sections, especially in teased ones, it is possible to distinguish, besides the fat-cells and blood-capillaries, a honeycombed matrix of connective-tissue, which consists of flattened, nucleated connective-tissue cells, attached to a plexus of very thin fibre-bundles derived from the interlobular septa.

The successful injections make it highly probable that each of the above intercellular lymphatics is bounded by the fat-cell on the one side, and the intercellular honeycombed connective-tissue on the other. Thus an analogy would be established between the ultimate lymphatics in the adipose tissue of the skin and those in the nerve trunks (Key and Retzius, Studien in d. Anatom d. Nervensystems, &c.), in the latter the lymphatics between the neighbouring nerve fibres being separated by the endoneural connective-tissue.

I need hardly add here of how great importance in physiological and pathological respects this intimate relation between the adipose tissue of the skin and the absorbents is, since that tissue plays a very prominent part in the economy of the body in health and disease.

In my Anatomy of the Lymphatics, Part I., Chapter II., I have shown that in its formation the fat-tissue of the serous membranes has an intimate relation with the ultimate lymphatics of those parts, the fat-cells being formed from connective tissue-cells (Flemming) situated in the rootlets of the lymphatics, *i.e.* in the lymph-canalicular system. What I have now described of the anatomical distribution of the intercellular lymphatics in the fat-tissue of the skin bears this out in a very striking degree.

C.—*The Lymphatics of the Sweat Glands.*

25. The sweat glands also possess their own minute lymphatics. These are very distinct in preparations of the human skin successfully injected with asphalt-benzole; they are taken up by the lymph-vessels of the surrounding tissue described above, *i.e.* those belonging to the plexus of lymphatics of the connective-tissue ground substance. In Fig. XV. are seen the *lymph-clefts* situated between the coils of the sweat-

gland tube and the connective-tissue separating the former. The relation of the lymph-clefts to the coils of the gland tube on the one hand, and to the connective-tissue on the other, appears to be the same as that described of the fat-tissue, viz., the lymph clefts do not possess their own proper wall of endothelium. Along the duct of the sweat gland, while passing through the corium, lymph-clefts may also be traced for a longer or shorter distance, as is shown in horizontal sections in which the ducts are cut transversely, or nearly so. The above lymph-clefts surround sometimes half or even more of the circumference of the duct, so that this latter appears almost invaginated by the lymphatic.

D.—*The Lymphatics of the Hair-Follicles and Sebaceous Glands.*

26. The hair-follicles possess, according to Teichmann, G. and F. Hoggan, no lymphatics. Neumann describes lymphatic vessels which surround the hair-follicles as the proper vessels of these latter; but I recognise in his description and illustration (l.c., Fig. 5, Plate III.), merely lymphatics of the general connective-tissue ground substance.

After a great many negative results, I have succeeded in a few instances in the human scalp as well as in that of the dog, in injecting the ultimate lymphatics of the hair-follicles. Both in the asphalt-benzole injections, but especially in those with Berlin blue, I have ascertained that the lymphatic vessels and interfascicular lymph-spaces of the connective-tissue surrounding the hair-follicle bear an intimate and, I need hardly add, important relation to the latter.

The fibrous coat of the hair-follicle, that is, the hair-sac proper, contains, both in human hair as well as in that of the dog, more or less continuous lymph-channels, which are in communication with the plexus of the lymphatic vessels of the corium, and with the interfascicular lymph-spaces around. In a longitudinal section these lymph channels are well seen in close neighbourhood to the outer root-sheath of the hair. *See* Fig. VIII. In transverse sections it is ascertained that the lymphatics of the hair-sac *surround like a sinus the outer root-sheath. See* Figs. X. and XI.

In these last two figures, which represent hair-follicles of the scalp of the dog, the lymphatics of which had been injected with Berlin blue, it is seen with great distinctness that the injection material is contained not only *in the lymphatic channels and spaces of the hair-sac and its surroundings*, but that it *penetrates also into the root-sheath.* The injection material passes directly from the lymph-sinus around the root-sheath *into the interstitial or cement substance between the epithelial cells*, and what is still more remarkable and interesting, the injection material, having penetrated up to the inner root-sheath, accumulates there *in a disinct layer or space between this and the outer root-sheath, and also between the former and the hair itself.* It is very probable that it reaches this latter in the same way as the former, viz., through the cement substance of its (*i.e.* the inner root-sheath's) scales. In the above case of the skin of the new-born child, whose lymphatics contained smaller and larger clumps of sebum, there existed most distinct signs of this latter substance being accumulated between the hair itself and the inner root-sheaths; further, between this and the outer root-sheath minute bright sebum-particles were to be met with in the cement-substance between the epithelial cells of the outer root-sheath, and were seen accumulated in the lymph-channels of the hair-sac. This is, then, clear evidence that the paths followed by the above Berlin-blue injection are the real ways of absorption, that is, are not produced artificially, since in the case of the sebum-

absorption above referred to, it cannot be denied that the absorption had taken place during life.

The anatomical connexion of the lymphatics of the connective-tissue with the interstitial substance between the epithelial cells of the root-sheath is in perfect agreement with what is now well established for the mucous membranes, glands, serous membranes, &c. And I would again refer in this respect to the researches of Arnold, Thoma, myself, Küttner, Sikorsky, Watney, and others.

Just as I have shown for the epithelium lining the bronchi and alveoli of the lung and for the endothelium covering the serous membranes, so also in the case of the stratified epithelium forming the outer root-sheath of the hair we find in the interstitial cement substance, branched, nucleated connective-tissue cells anastomosing with the connective-tissue cells of the surrounding hair-sac.

That this connexion of the lymphatics of the hair-sac with the space between the hair and the inner root-sheath is of the utmost physiological and pathological interest is obvious. There can be no doubt that, in the absorption of fluid and formed matter from the outer skin the lymphatics of the hair-follicles play a conspicuous part, and the practical experience of the efficiency of medicaments, especially those suspended in fatty matter rubbed into the skin, by which they are well pushed into the depth of the hair-follicles, receives hereby its theoretical explanation.

R. Fleischer, Erlangen 1877, and quite recently in Virchow's Archiv., vol. 79, p. 458, still questions the positive results obtained by Lassar (Virchow's Archiv., vol. 67, 1) as to the passage of oily matters rubbed into the intact skin of the rabbit, through the absorbents into the internal organs, for he (viz., Fleischer) maintains that such passage does not take place except through the injured skin.

My results above described clearly show that the anatomical facts are in favour of Lassar's assertions.

27. Whether a similar intimate connexion exists between the lymphatics around the sweat gland (both duct and coiled tube) and the interstitial cement substance of its epithelium and the inner cavity of the tube, is very probable, but I have not been fortunate in showing this. Inunctions with pigment suspended in fatty matter would, I have little doubt, yield positive results.

28. As regards the lymphatics around the sebaceous glands and the muscle of the hair, I am able to record positive results, against the negative ones of Teichmann and G. and F. Hoggan. Neumann's assertions as to the existence of lymphatics of these organs are of the same value as those referring to his lymphatics of the fat-tissue and the hair-follicles, viz., they belong merely to the surrounding connective-tissue.

In preparations of the human scalp, the lymphatics of which had been successfully injected with asphalt-benzole, the alveoli of the sebaceous glands are seen surrounded for a larger or smaller part of their circumference by *lymphatic spaces and sinuses* which are connected both with lymphatic vessels and with the interfascicular lymph-spaces of the connective-tissue.

Elongated lymphatic clefts are also seen between the bundles of the arrector muscle, similar to those to be observed in the unstriped muscle-tissue generally.

In Fig. XVA. the lymphatics of a sebaceous gland and arrector pili of the scalp of a new-born child are seen injected with asphalt-benzole.

II.—The Lymphatics of the Skin of the Frog.

29. Owing to the great abundance of glands in the skin of the frog, and owing also to the importance in the process of absorption of this organ, I have investigated its lymphatics also. But there were very great difficulties to overcome. What I intended to do was to demonstrate by a good injection the arrangement and distribution of the lymphatics. The method of puncture was here out of question, for it was impossible for me to obtain a canula of the necessary fineness. It occurred to me that Budge's method of forcing by pressure (of air, water, or mercury) the injection material into the lymphatics as practised by him in the case of the lymphatics of the cartilage and bone (l.c.) might be productive of good results. My expectations were soon realised. The method I follow is this: in a decapitated frog I cut off the anterior extremities and enucleate the trunk from its skin, after severing the septa between the cutaneous lymph-sacs of the chest, abdomen, and dorsum; I then place a ligature round the root of the trunk and cut this latter off close to the ligature. I have now a preparation that consists of the entire lower extremities and the empty skin of the abdomen, chest, and dorsum. This quasi-bag is tied round the mouth of a small funnel specially provided there with a groove, and the tube of the funnel is fixed in a retort-holder, so that the preparation is hanging downwards. The tube is a long glass tube, and is connected with the injection-bottle containing the injection fluid. This is pressed into the funnel by a mercury pump.

A pressure of 2 to 2½ inches being applied, and a 2 to 4 p.c. solution of Brücke's Berlin blue being used, it is found that the injection soon forces its way into the cutaneous lymph-sacs of the lower extremities, without however penetrating into the lymphatics of the skin itself. But on the inside of the thigh next the median line, and in the neighbourhood of the anus, *i.e.*, in those parts which are in the common frog conspicuous by their thickness owing to warty prominences of the cutaneous gland-sacs, a beautiful injection appears which gradually increases in breadth and intensity, but does not penetrate anywhere into the lymphatics of the ordinary thin skin of the surrounding parts. Asphalt-benzole yielded no results.

30. Looking at the injected skin with the unaided eye, there appears a beautiful mosaic or network of blue lines, each of its meshes corresponding to a wart. Under a lens, or still better, under a low power of the microscope, it is seen that what with the unaided eye appears as a single line of the mosaic, is a dense plexus of lymphatics.

In Figs. I., II., and III. I have given representations of these lymphatics as seen under the microscope.

There is a deep plexus of large vessels connected with a much denser plexus of fine vessels belonging to the superficial part of the skin. These latter vessels are of various sizes and of a very irregular outline. Fig. III. shows them very well as seen under a higher power. The plexus of the finer vessels is interrupted by the cutaneous glands, as is noticed in Fig. III. But there exist special lymphatics surrounding a smaller or greater section of the circumference of the gland-sacs; these latter, viz. gland-sacs, are shown only in outline in this figure.

The lymphatics destined for the glands are apparently terminating with a cœcal extremity; and there are also other vessels of the general plexus which appear to terminate in a cœcal manner in the connective-tissue of the skin.

III.—The Lymphatics of the Conjunctiva.

31. According to Schmid (Die Lymphfollikel d. Bindehaut Wien, 1871), the lymphatics of the conjunctiva form a superficial and deep network connected with one another by many short branches. The vessels of the superficial network are very fine, and some of them terminate in the tissue with a pointed or cœcal extremity. The vessels of the deep plexus are possessed of valves. The superficial network is densest at the limbus, and less dense in the conjunctiva fornicis than in the conjunctiva palpebræ.

I have investigated the lymphatics of the eyelids of the new-born child and of the rabbit. The best preparations were here also obtained with the asphalt-benzole injection.

32. The lymphatics of the skin are to be distinguished from those of the conjunctiva palpebræ.

Injecting into the subcutaneous tissue of the eyelid, it will be seen that the lymphatics of the skin become injected up to the outer free margin: the injection does not apparently pass into the lymphatics of the conjunctiva.

In making sections through such an eyelid it will be found that the distribution of the lymphatics of the corium differs in no way from those of other parts; the plexus of the subcutaneous lymphatics is conspicuous by the size of the vessels and by their large saccular dilatations; this plexus takes up lymphatic-vessels situated between the bundles of the sphincter orbicularis. These vessels form a plexus, and are possessed of valves. They extend up to the dense fibrous tissue which forms the tarsal plate.

In vertical sections through the eyelid there are seen occasionally, but rarely, fine lymphatic vessels *passing through the tarsal plate* so as to join the lymphatics of the conjunctiva palpebræ.

The chief communication is, however, established at the margin of the eyelid, where the lymphatic vessels of the corium anastomose with the vessels belonging to the conjunctiva palpebræ proper, situated behind the tarsal plate, *i.e.*, between the layer of the Meibomian glands and the epithelium covering the free surface of the conjunctiva.

These results are in perfect agreement with those obtained by E. Fuchs in Medic. Centralblatt, 1878, N. 28.

33. To obtain a good view of the lymphatics of the conjunctiva palpebræ and fornicis, the canula must be inserted into the proper tissue of the conjunctiva palpebræ.

When successful, the inspection of the surface with a lens reveals an exceedingly dense network of lymphatics situated very near the surface. In the human conjunctiva palpebræ these vessels are very fine and the plexus extends to the free margin of the eyelid. The arrangement of the vessels is very characteristic, for about the first third, that is, the one nearest the free margin, they form a dense network with small and polyhedral meshes, in the second third the meshes become slightly elongated, but in a more or less oblique direction, the vessels assuming a tendency to run towards the angle of the eye, and in the last third, that is the one nearest the fornix, the meshes become still more elongated and have an almost horizontal position, the vessels following a course very nearly parallel to the margin of the lid. They join everywhere the plexus of the lymphatics of the fornix. These latter appear as a plexus of fine vessels situated superficially and one of large deep vessels; most vessels of both plexuses follow a course more or less parallel to the margin of the lid, *i.e.*, longitudinal towards the angle of the eye, as mentioned above.

In Fig. XVIB. will be found a representation of these lymphatics as seen in the birds-eye view under a lens.

In horizontal and vertical sections through the eye-lid the lymphatic vessels of the conjunctiva palpebræ extend from near the epithelium through the whole thickness of the conjunctiva.

The layer next the Meibomian glands contains large vessels (Colasanti) with valves, and these may be considered as the deep lymphatics.

Fuchs also injected lymphatic sinuses around the alveoli of the Meibomian gland.

In the plexus of the superficial lymphatics of the fornix, and also in that of the conjunctiva palpebræ nearest the fornix, there are found some vessels possessed of valves and corresponding saccular dilatations, but most of them are without valves.

Injecting in the same manner the conjunctiva of the rabbit it will be observed that in the conjunctiva palpebræ the blood capillaries and veins often become injected. Viewed with a lens or under the microscope the deep lymphatics with valves are also well seen.

The lymphatics of the conjunctiva fornicis come out with great distinctness, both those of the superficial as well as those of the deep parts.

In Figs. XVI., XVIA., and XVII. these relations are well shown, may be and easily understood from the description of the figures accompanying them.

The fact of the blood capillaries and veins becoming injected would lead one at once to say that the passage of the injection matter into the blood-vessels is due to an injury of the blood-vessels by the cannula. But on careful examination I failed to detect this, but on the other hand I have found what appears to be a direct anastomosis of minute veins with lymphatics.* This is, however, a point of so great an importance that in order definitely to express an opinion, a greater number of observations and a more minute examination than I have been able to make would be required.

I have good reason to believe that there exists an indirect communication between the blood-capillaries of the conjunctiva palpebræ of the rabbit and its lymphatics through the lymph-canalicular system (v. Recklinghausen). In Fig. XVIIA. I have given a representation of an exceedingly fine injection with asphalt-benzole of the lymph-canalicular system (interfascicular lymph-spaces), leading directly on the one hand into a lymphatic and on the other into the capillary blood-vessels.

That the anastomosis of the lymph-canalicular system with the blood-capillaries appears here so patent and extensive may be owing to abnormally delicate capillaries.

It is possible that in the rabbit the interstitial cement substance of the endothelial wall of the capillaries of the conjunctiva palpebræ is more liable to give way and to allow the injection material to pass from the lymphatic rootlets into the blood capillary.

IV.—The Lymphatics of the Oral Cavity and Pharynx.

35. Teichmann (l.c.) and subsequent writers quoting his work, describe the lymphatics of the oral cavity and pharynx as being arranged similar to those of the skin, as a superficial plexus of fine

* From an inspection of Figs. XVI. and XVII. such a communication is in the highest degree probable v. Recklinghausen observed a similar passage of the injection matter from the lymphatics into the blood vessels. Waldeyer considers it due to an artificial rupture.

vessels and a deep one composed of large vessels with valves. The superficial plexus is the denser of the two, and is in connexion with the cœcal lymphatics belonging to the papillæ.

The observations which I propose to describe here refer to the lymphatics of the lips, buccal mucous membrane, the palate and pharynx, and the tongue They were obtained by injecting into the mucous membrane, as near as possible to the surface of these different regions, asphalt-benzole or Berlin blue in precisely the same manner as was described on a former page in connexion with the skin.

The results of these injections confirm in a general way the observations of Teichmann, but extend them in some special details; the relation of the lymphatics of the connective-tissue ground-substance to the epithelium of the surface, the lymphatics of the glands and the muscular bundles, are points which in Teichmann's work have not been sufficiently considered, but which, as I shall be able to show, deserve special attention.

As in the case of the skin, so also in that of the mucous membranes, it is necessary to consider the lymphatics as forming different groups, such as the lymphatics of the ground-substance of the mucous membrane; those of the fat-tissue, where such is present; those of the glands, mucous and serous; and finally, those of the muscular tissue.

The Lymphatics of the Ground-Substance.

36. In a successful injection into the mucous membrane of the lips of the mouth, it will be found that there appears a very dense and beautiful network of fine lymphatics situated close to the surface. When examined under a lens, it will be seen that the network is very dense, and that the vessels are either straight or slightly wavy and irregular. A few larger vessels with distinct valves may be seen in the depth; the direction of the course of the efferent trunks is away from the free margin of the lip, in the parts near the middle line straight towards the gums, at the side towards the mucous membrane of the angle of the mouth.

When injecting into the part situated between the cutis and mucous membrane of the lip, it will be found that the injection material passes much more frequently into the lymphatics of the mucous membrane than into those of the cutis. But in every injection into the cutis of the one side of the lip the injection does not readily pass beyond the middle line, that is, from one side into the other. Injecting, however, the lymphatics of the mucous membrane, no such demarcation between the two sides can be observed.

37. The injection of the buccal mucous membrane and of that of the palate and pharynx shows also—these organs being examined in the fresh state in the bird's-eye view—a beautiful and dense network of fine lymphatics, the meshes being of a more or less uniform polyhedral shape.

38. In the tongue, however, the aspect of the lymphatics, as seen in the bird's-eye view of the fresh organ with the unaided eye or with a lens, greatly differs from the above organs, owing of course to the presence of the papillæ. On the dorsal part of the tongue there is found a network of fine lymphatics, whose meshes correspond to the papillæ; they are naturally larger for the fungiform than for the filiform papillæ.

The lymphatics of the tissue of the papillæ themselves are not visible under these circumstances.

Owing to the peculiar change of the surface at the margin of the tongue, viz., the papillæ becoming arranged in more or less distinct transverse

rows, the shape of the network of the lymphatics undergoes a corresponding variation, *i.e.*, its meshes become elongated in a transverse direction.

At the margin of the tongue we notice numerous efferent vessels passing from the dorsum on to the lower smooth surface; they are more or less parallel with one another.

At the base of the tongue the arrangement of the lymphatics changes from a uniform network of fine vessels into one with elongated meshes. There is an exquisitely dense network of very fine vessels surrounding each circumvallate papilla, as shown in Fig. XXII.

In man and in animals I have always found a plexus of large and fine lymphatics running from the dorsum of the tongue towards the base of the epiglottis, such as is shown in Fig. XXII. The vessels anastomose under acute angles and increase in size as they approach the epiglottis; their valves are very conspicuous.

The dense network of the lymphatics of the mucous membrane of the gums and soft palate and tongue has already been injected by Sappey.

39. Examining a successfully injected and hardened lip, palate, or mucous membrane of pharynx in vertical and horizontal sections under the microscope, it is seen that the lymphatics are present in, and extend through all parts of the mucous membrane more or less uniformly from the epithelium of the surface down to the deep tissue that contains the glands, fat, muscle, &c. The vessels are connected everywhere in plexuses, but there are in the superficial parts a good many vessels that appear to terminate freely in the tissue (*see* below).

Many vessels run in a horizontal direction, and they can be therefore better seen in horizontal than in vertical sections. Those belonging to the superficial parts of the mucous membrane are fine as compared with those of the deeper parts, and they run a more or less straight or wavy course. *Everywhere the papillæ possess their own lymphatics in the form of single or compound loops connected with the superficial lymphatics.* They penetrate more than half or two-thirds into the tissue of the papillæ. These papillary lymphatics are best seen in the human lip and soft palate, owing to the very great length of the papillæ; but they are present also in those of other parts in man, dog, and rabbit. The lymphatics of the deeper parts are distinctly possessed of valves; such valves are not easily seen in the more superficially seated lymphatics, although in some isolated instances there are indications of their presence in the shape of saccular dilatations of the vessel.

The wall of the lymphatics of the superficial layers is irregular in its outline, being apparently possessed of more or less numerous pointed processes (*see* below).

The lymphatics of the loose submucous tissue are considerably larger and possessed of valves; they communicate freely with the plexus of the large lymphatics of the muscles and the fat-tissue.

40. The relation between the lymphatics of the mucous membrane and the interfascicular lymph-spaces of the connective-tissue ground substance is a very intimate one, and is exactly the same as that described of the connective-tissue of the skin. It is therefore unnecessary to minutely describe this relation again, and I will merely refer the reader to Figs. XX. and XXI. The irregular outline of the wall of some of the lymphatics is due to the lumen of the vessels being in open communication with the interfascicular spaces of the surrounding tissue.

41. An important and intimate relation exists between the most superficial lymphatics and the epithelium of the surface.

This relation is of the same nature as the one I have shown to exist between the epithelium constituting the outer root-sheath of the hair-follicles and the surrounding lymphatics, viz., *a direct passage of injection material from the lymphatics*,—both the lymphatic vessels and sinuses as well as the interfascicular lymph-spaces—*into the interstitial or cement-substance between the epithelial cells.* That there exists a direct connexion between the lymph-spaces of the most superficial layer of the mucosa, including the papillæ, and the interstitial cement-substance of the epithelium, can be ascertained in every thin section of a well-prepared mucous membrane: it will be found on careful examination that the spaces between the fine connective-tissue bundles,—some of which correspond, as is shown in injections, to lymphatic vessels—are distinctly prolonged into the interstitial substance of the epithelium. This is still more conspicuous in slight inflammatory conditions, when the lymph-spaces are enlarged and the interstitial substance is much increased in amount. The branched cells situated in the interstitial substance of the epithelium, mentioned on former occasions, are also here in connexion with the connective-tissue cells situated in those lymph-spaces.

In Fig. XIX. is represented a horizontal section through the most superficial parts of the mucous membrane of the lip of a rabbit, and it is noticed here, that the interstitial substance of the epithelium, which includes here as well as in many other mammals *pigment granules* and *pigmented* branched cells, contains the injection matter (Berlin blue), and that this passes directly into the interfascicular lymph-spaces and the most superficial lymphatic vessels.

The very same condition exists everywhere in the oral mucous membrane and pharynx as regards the interstitial substance of the stratified epithelium of the free surface and the superficial lymphatics,—a condition which I need hardly say is of great physiological importance. This relation can be studied in sections of simple uninjected preparations as well as in successfully injected specimens, and it is borne out by the observations of Toma and Arnold and others as regards the relation existing between the interstitial substance of the epithelium of the mucous membrane of the frog's mouth with the tissue of the latter.

42. In vertical sections through the injected tongue we see, under the microscope, a very rich plexus of lymphatics situated in the thin mucous membrane; these vessels are very irregular in size and outline, and some of them possess valves. Their course is chiefly parallel to the surface, and both in longitudinal as well as in transverse sections through the tongue a great many vessels are seen cut longitudinally.

Sappey describes fine vessels which enter the papillæ and then form a network. I can fully confirm this, for I find that each papilla filiformis and fungiformis (which, as is well known, are again possessed of minute secondary papillæ) of the human tongue contains its own network of lymphatics. *From the network we see lymphatic vessels ascending the secondary papillæ* and either terminating in a cœcal extremity or in a loop.

In animals (dog, rabbit) this is so far different, that the filiform papillæ are mostly simple, not compound, but in the fungiform papillæ the same relations exist as in those of the human tongue.

43. The papillæ circumvallatæ of the human tongue contain a very great number of lymphatics. They are all fine vessels connected into a network, which does not show any special richness at the base where the taste-goblets are found; on the contrary the vessels are if anything less numerous at that part than at the rest of the "papilla."

In the papilla foliata of the rabbit each of the ridges, which constitutes this organ, and as is well known, corresponds to the "papilla" of a circumvallate papilla drawn out into a fold, contains at its basis rows of taste-goblets; we find here so far a variation of the above arrangement of the lymphatics, that from the superficial network there generally *ascends in the centre of each of the folds a cleft-like or sinuous lymphatic* which terminates in a cœcal extremity near the top of the fold, but does not reach as high as the blood-vessels. In some folds small branchlets may be found to come off at the basis from the central vessel; they then ascend towards the taste-goblets. It is not possible to ascertain whether they stand in any definite relation to the latter.

44. At the root of the human tongue where the mucous membrane contains masses of adenoid tissue, either diffuse or as more or less well-defined lymph-follicles, this arrangement of the lymphatics so far suffers an alteration that their plexuses become as it were interrupted by the lymph-follicles.

These latter appear surrounded by saccular lymphatics, but amongst the diffuse masses of adenoid tissue may also be seen lymphatic vessels.

In both instances the endothelial wall of the lymphatic is *directly continuous with the reticulum of the surrounding adenoid tissue*, in the same manner as I described it of both the serous membranes and the bronchial walls in Part I. and II. of my Anatomy of the Lymphatic system.

35. The lymphatic vessels of the deeper parts of the tongue, that is, of the connective-tissue separating the muscle-bundles of the tongue, are exceedingly numerous, very large, possessed of valves, and anastomosing into a rich plexus. Their efferent trunks run in company with the large blood-vessels, forming around these a beautiful plexus.

The arterial branches of the muscular tissue of the tongue as well as those entering the mucous membrane are generally accompanied by one or two lymphatic vessels; in some instances these latter appear like sinuses, *invaginating the whole or the greater part of the circumference of the blood-vessel.*

46. The arrangement of the lymphatics of the fat-tissue, in the lip, in the submucous tissue of the soft palate, especially at the base of the tongue, being in all respects identical with that described of the fat-tissue of the skin, does not require any special description. The connexion of the lymphatic vessels situated in the connective-tissue septa between larger or smaller groups of fat-cells with their ultimate radicals between the individual fat-cells is very distinct in parts, where the lymphatics have been successfully injected. The root of the tongue of the child, rabbit, and dog have yielded the best preparations.

47. The mucous and serous glands in the root of the tongue, as well as the mucous glands in other parts of the oral cavity and pharynx, possess their own lymphatics. The tongue, after its lymphatics have been injected with Berlin blue, yields very beautiful specimens, if hardened and the sections obtained from it stained in picro-carmine. There can be no doubt whatever, that just as in the case of the sweat glands of the skin, so also in that of the mucous and serous glands, *the lymphatic vessels of the surrounding connective tissue anastomose with sinuous-like clefts surrounding more or less of the circumference of the gland tubes or alveoli.* These lymph clefts are situated between the membrana propria of the alveoli and the thin connective-tissue lamellæ separating these latter.

In Fig. XXIII. these interalveolar lymph-clefts of a portion of a serous gland in the root of the tongue of the rabbit are well shown.

Both the ducts of the mucous and serous glands possess their own lymphatics; they appear as lymph-spaces extending along the duct for a longer or shorter distance, and in many cases either completely invaginating it, as in Fig. XXIV., or only partially so, as in Figs. XXV. and XXVI.

48. The relation of the lymphatics to the striped muscle-tissue, where it occurs in the mucous membrane itself, *e.g.*, in the lip, palate, and tongue, is a very intimate one.

We have already mentioned the large lymphatic vessels present in the connective-tissue septa between the bundles of the muscle-tissue; they are connected into a plexus whose vessels run chiefly in a direction parallel to the long axis of the bundles. These vessels may be appropriately called the *interfascicular* lymphatics. Just as is the case with the fat-tissue, so also here, the ultimate lymphatic radicles are lymph-spaces between the individual muscle-fibres. Each muscle-fibre of a bundle is surrounded by a lymph-space (*see* Fig. XXVII.), and this latter is consequently contained between the sarcolemma of the muscle-fibre and the connective-tissue separating the individual muscle-fibres, *i.e.*, the endomysium. These lymph-spaces may justly be termed the *intrafascicular* lymph-clefts. The lymphatics, therefore, existing in the bundles of the striped muscle-fibres are identical with these of the nerve-bundles as described by Key and Retzius.

49. We have finally to mention the existence of continuous lymph-spaces in the sheath of the nerve-bundles and the capsule of the ganglia, existing in the connective-tissues both between the lobules of the glands and between the bundles of the muscle-fibres in the root of the tongue. These spaces correspond to the perineural lymph-spaces described by Key and Retzius of the cerebro-spinal nerves in general.

EXPLANATION OF FIGURES.

Fig I.

The deep plexus of the lymphatics of the frog's skin viewed from the surface under a low power, magnifying about 25. Injected with Berlin blue.

Fig . I.

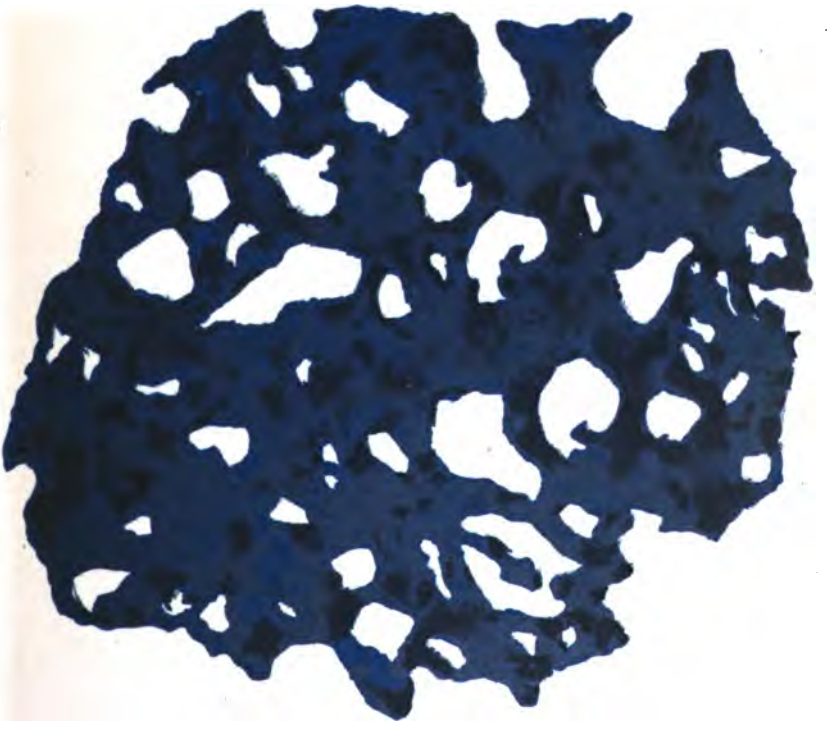

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Fig. II.

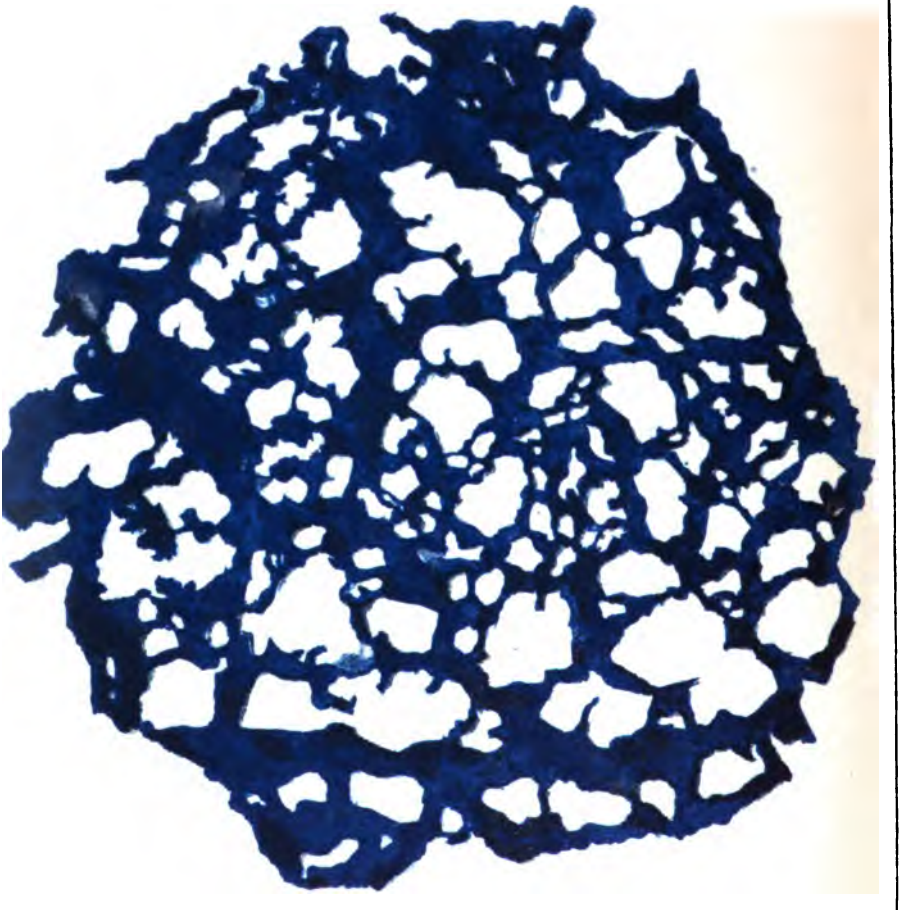

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FIG. II.

The superficial plexus of the lymphatics of the frog's skin as seen under a low power.

FIG. III.

The superficial plexus seen under a higher power, about 300.

b. Large vessels.

c. Minute lymphatic channels around the glands.

g. The cutaneous glands.

Fig. III.

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FIG. V.

From a horizontal section through the scalp of dog, showing the lymphatics of the layer 1 of figure IV., as seen from the surface. M. p. about 60.

m. Mouths of hair-follicles.

Fig. V.

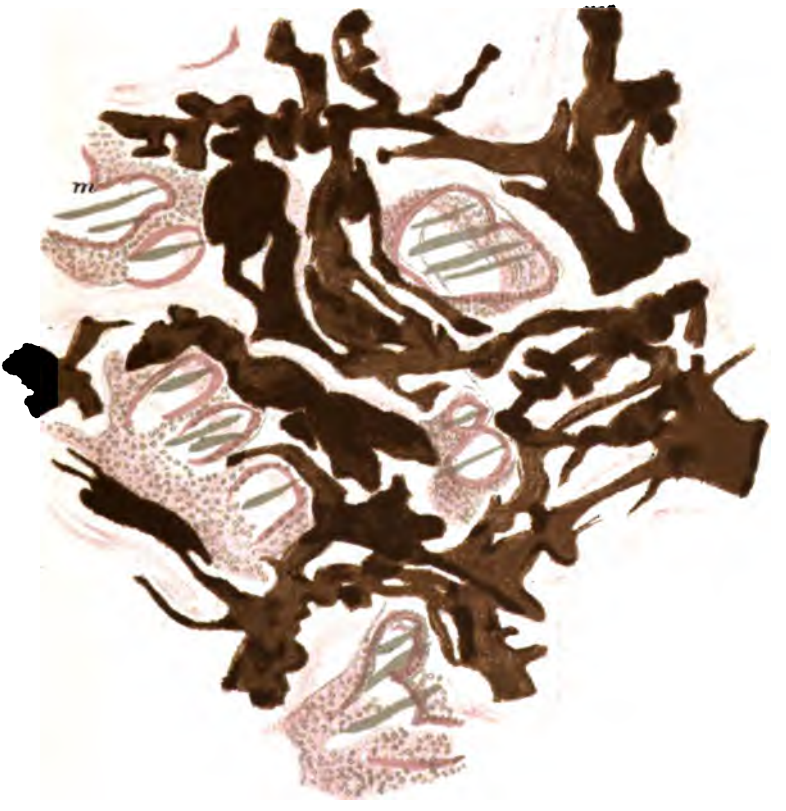

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Fig. VI.

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FIG. VI.

A similar view of layer 2. M. p. about 60.

c. Trabeculæ of connective-tissue.

h. Hair-follicles and sweat-gland tubes in transverse section.

FIG. VII.

A similar view of the lymphatics of layer 3. M. p. about 350.

f. Fat-cells.

c. Fibrous connective-tissue separating the fat lobules.

t. Large interlobular lymphatic vessel taking up the intercellular lymphatics.

Fig. VII.

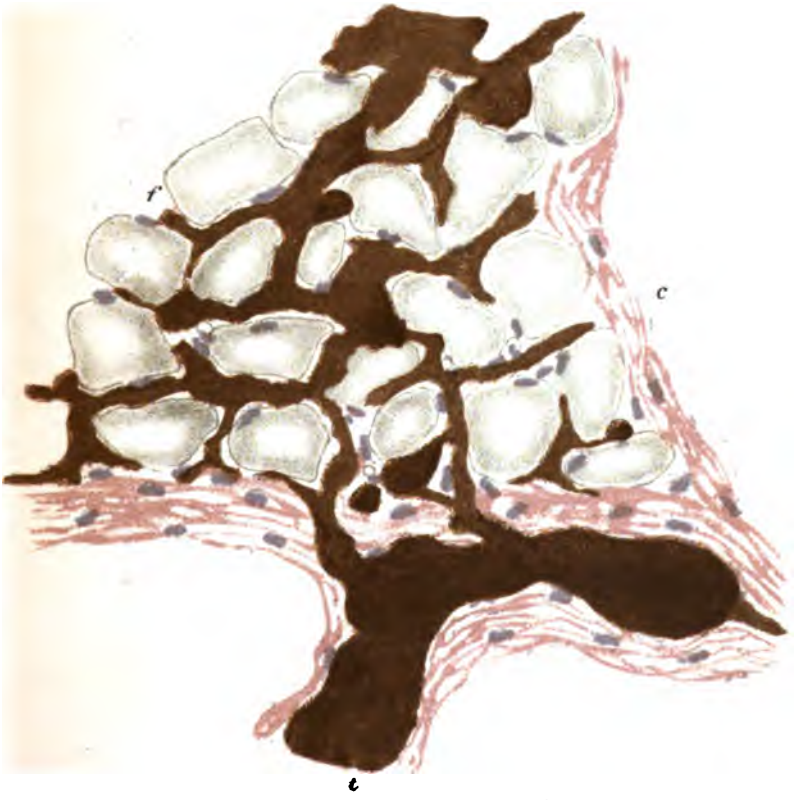

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Fig. VIII.

Fig. IX.

Dangerfield. Lith. 22. Bedford St. Covent Garden.

FIG. VIII.

From a vertical section through the scalp of dog, the lymphatics of which had been injected with asphalt-benzole. M. p. about 90.

h. Hair-follicle.

l. Lymphatics of its fibrous sac.

c. Lymphatics of the surrounding connective-tissue of the corium.

FIG. IX.

From a horizontal section through the corium of the scalp of dog, the lymphatics of which had been injected with Berlin blue, showing the interfascicular lymph spaces of the connective-tissue of the corium.

l. In longitudinal.

t. In transverse and oblique view. M. p. about 350.

Fig. X.

From a horizontal section through the scalp of dog; the lymphatics had been injected with Berlin blue. M, p. about 180.

h. Hair-follicle.

l. Lymph spaces around it.

v. Lymphatic vessels of the surrounding connective-tissue.

s. Sweat-gland tube, also surrounded by lymph-spaces. (See the text.)

Fig. XI.

From a similar preparation as Fig. X. M. p. about 450.

h. The hair and its outer root sheath.

l. Lymphatic vessel of the surrounding connective-tissue.

s. Lymph space of the hair sac. (See the text.)

Fig. XI.

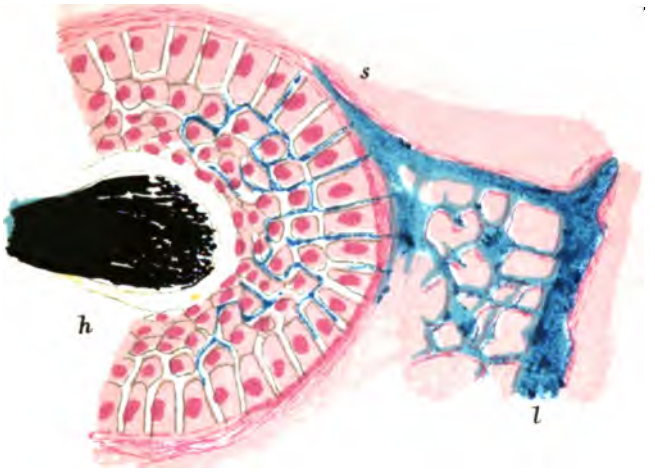

Fig. X.

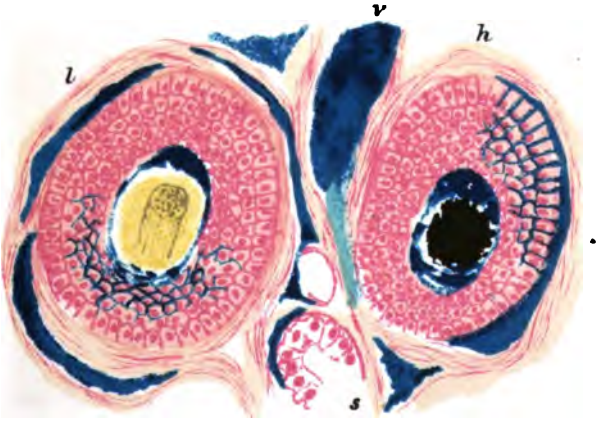

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Fig . XII.

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FIG. XII.

From a horizontal section through the scalp of a new-born child. Low power. Showing a few lymphatics of the corium.

t. Lymphatic trunks containing clumps of fat. (See text.)

FIG. XIII.

From a horizontal section through the skin of the cheek of a child; the lymphatics had been injected with asphalt-benzole.

c. Connective-tissue separating the fat-lobules, and containing a dense plexus of minute lymphatics.

f. Fat-cells and between them the intercellular lymph-channels. M. p. about 100.

Fig. XIII.

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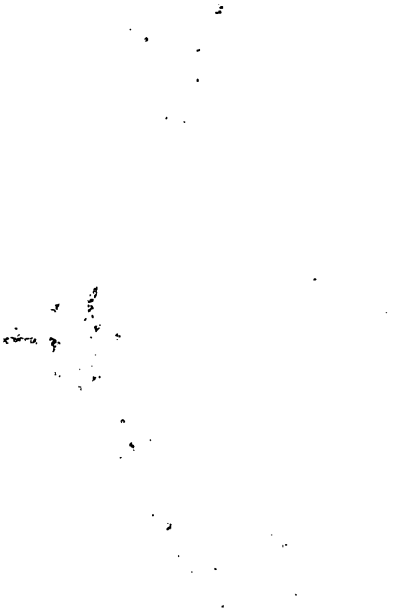

Fig . XIV.

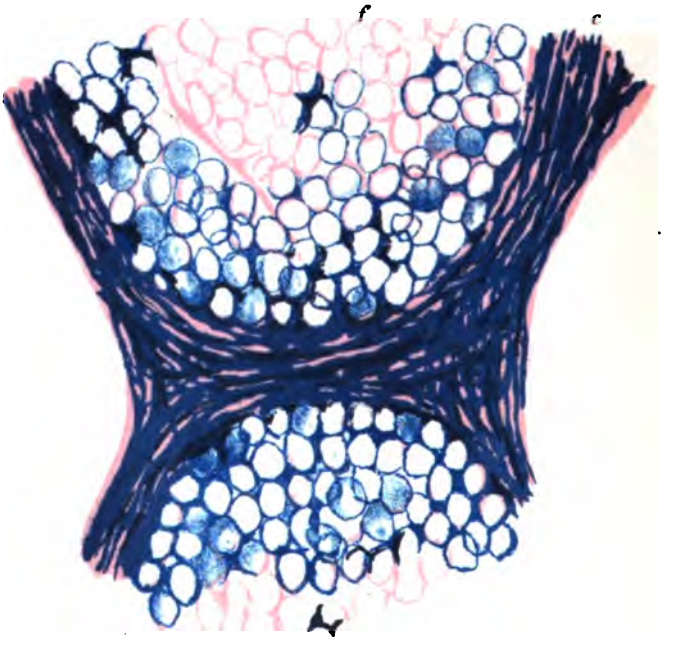

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FIG. XIV.

A similar preparation as Fig. XIII., but the lymphatics had been injected with Berlin blue. M. p. about 100.

c. Connective-tissue septa between the lobules of fat-cells.

f. The fat-cells.

Fig. XV.

From a section through the scalp of a newborn child. M. p, about 350.

c. Trabeculæ of the connective-tissue, cut in various directions.

s. Sweat-gland tubes cut in different directions.

l. Lymphatics taking up the lymph spaces between the gland-tubes, and between the trabeculæ of the connective tissue.

Fig. XVa.

From a similar preparation as the preceding Fig XV. M. p. about 450.

Showing the lymphatics of the connective-tissue matrix, of the musculus arrector pili and of the sebaceous-follicle.

d. Duct of the hair-follicle.

h. The hair.

s. Central part of the sebaceous-follicle.

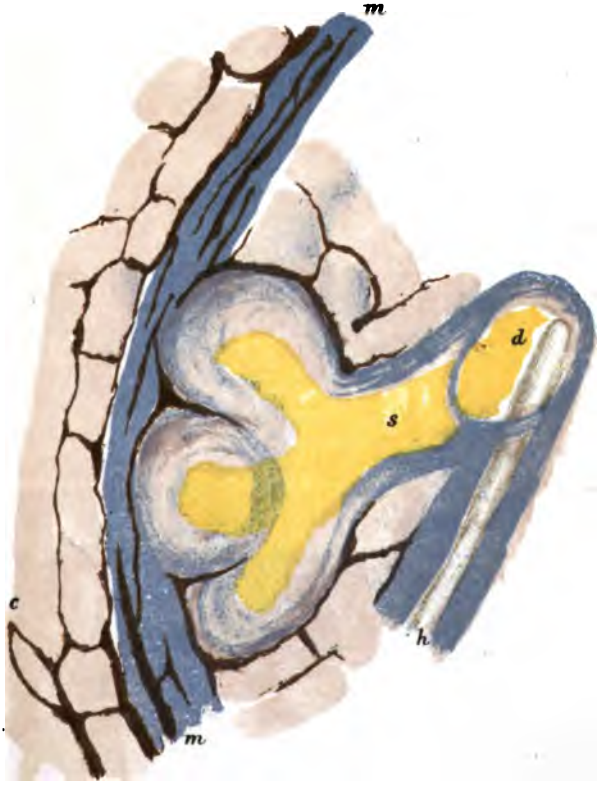
c. The surrounding trabeculæ of the connective tissue.

m. Musculus arrector pili.

The details of the structure of the sebaceous gland, hair-follicle, and muscle are not indicated.

Fig. XV.

Fig. XVA.



Dangerfield. Lith. 22. Bedford St Covent Garden

XVIA.

The plexus of lymphatic vessels of the conjunctiva fornicis of the rabbit, seen under a lens.

XVIB.

Surface view of the lymphatics of the conjunctiva of the lid of a newborn child, as seen under a lens.

A. The region of the conjunctiva palpebræ; the Meibomian glands are seen in outline extending from the lid margin.

B. The region of the conjunctiva fornicis.

Fig. XVI.A

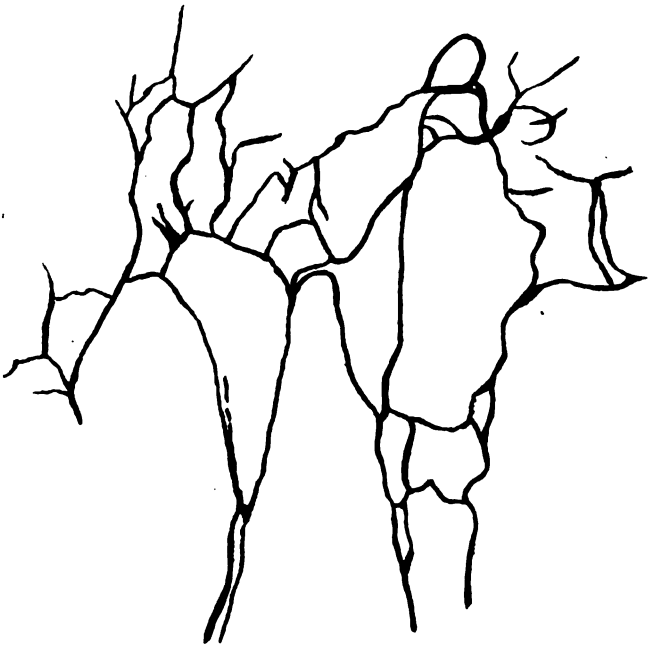
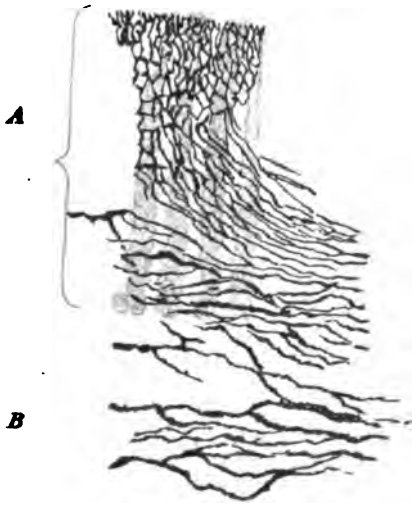


Fig. XVI.B



Dangerfield Lith. 22 Bedford St Covent Garden

Fig. XVII.

Dan.l Fearfield Lith 22 Bedford St Covent Garden

FIG. XVII.

Horizontal view of the conjunctiva fornicis of a rabbit. M. p. about 150.

l. Plexus of superficial lymphatics.

t. Large lymphatic trunks.

b. Blood-vessels.

XVIIa.

H orizontal view of the conjunctiva palpebræ of a rabbit. M. p. about 300.

b. Blood capillaries.

cs. Lymph-canalicular system.

l. A lymphatic vessel.

Fig. XVII A.

Dangerfield Lith. 22 Bedford St. Covent Garden.

Fig. XVIII.

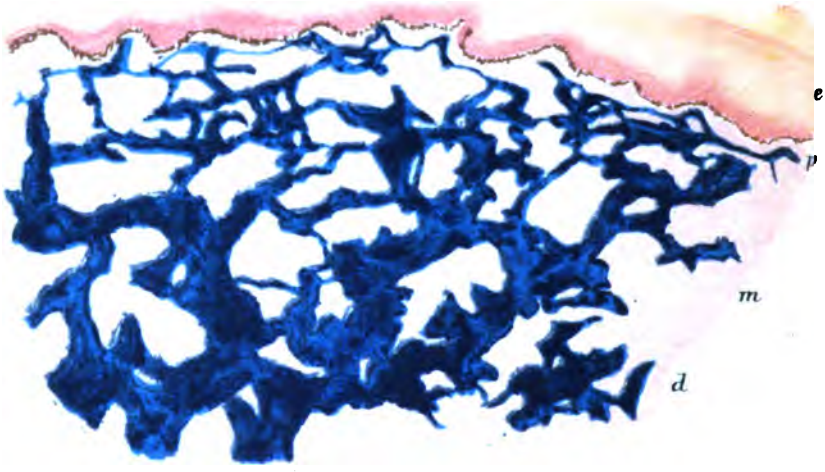
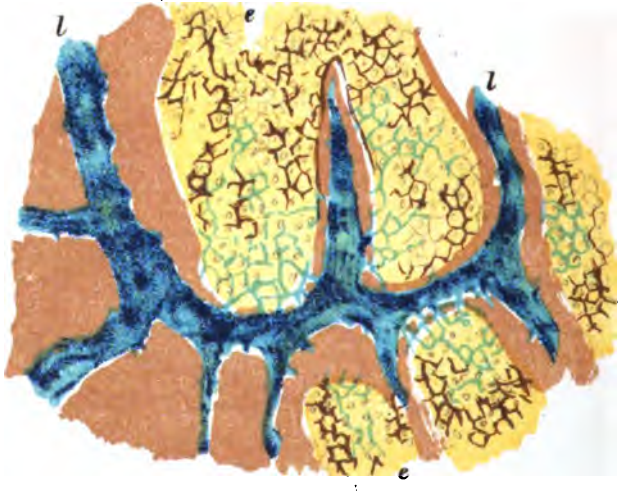


Fig. XIX.



Dandgefield. Lith. 22. Bedford St. Covent Garden.

Fig. XVIII.

From a slightly oblique section through the mucous membrane of the angle of the mouth of a rabbit. The lymphatics are injected with Berlin blue. M. p. about 60.

e. Epithelium: the individual cells are not indicated.

p. Papillary layer and the superficial lymphatics.

m. The middle layers of the lymphatics.

d. The deep plexuses of the lymphatics.

Fig. XIX.

From a horizontal section of a similar preparation as in Fig. XVIII. M. p. about 260.

e. The epithelium horizontally or obliquely cut. The interstitial cement substance between its cells contains pigment, and the injection has found its way into it from the lymphatics of the mucosa.

The lymphatic vessels of the superficial plexuses, *i.e.*, those situated in the papillary layer of the mucosa.

FIG. XX.

Lymphatics of the superficial plexus represented in Fig. XVIII. M. p. about 300.

Fig. XX.

Dangerfield Lith. 22 Bedford St. Covent Garden

Fig. XXI.

Daguerreotype. Lith. 22. Bedford St. Covent Garden.

FIG. XXI.

Similar lymphatics of the middle stratum. M. p. about 350. The lymphatics are everywhere connected with the interfascicular lymph-spaces, hence the numerous fine processes.

Fig. XXIIa.

Vertical section through the papilla foliata of the rabbit. The lymphatics are injected with Berlin blue. M. p. about 90. The section is stained with picro-carmine.

E. The epithelium of the surface.

g. The grooves between the taste-folds.

T. Taste goblets in rows.

D. The ducts of serous glands of the depth, not included in the drawing.

XXII.

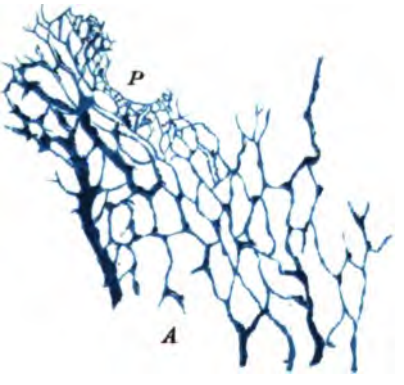
Plexus of lymphatics, in the root of the tongue of the rabbit, as seen under a lens.

A. Near the epiglottis.

P. Part of a circumvallate papilla.

Fig. XXII.ª

Fig. XXII.



Dangerfield. Lith 22 Bedford St Covent Garden

Fig. XXIII.

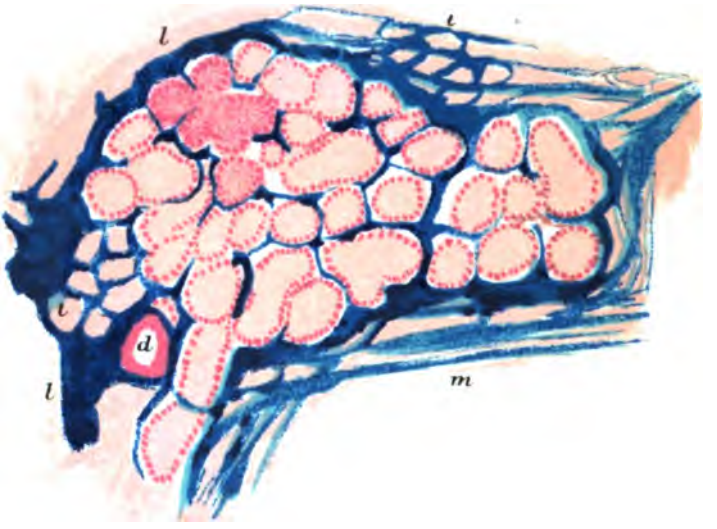
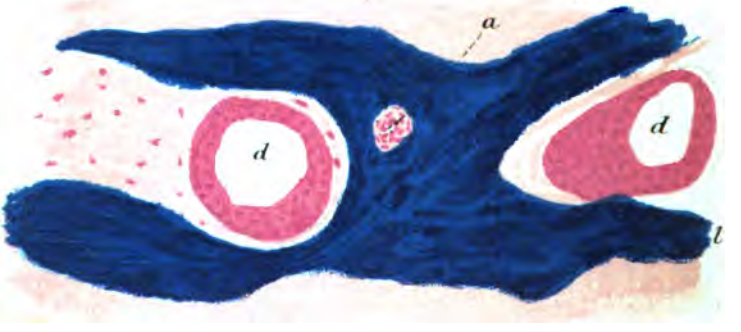


Fig. XXIV.



Dangerfield Lith. 29. Bedford St Covent Garden

Fig. XXIII.

A lobule of a serous gland from a similar tongue as in Fig. XXIIa. M. p. about 100.

l. Lymphatic vessels in connexion with the lymph-clefts between the gland-alveoli.

m. Lymph clefts between the muscular fibres, in a longitudinal view.

t. The same in transverse section; the structure of the muscular fibres is not indicated.

d. A duct in transverse section.

Fig. XXIV.

From a similar preparation as the preceding figure. M. p. about 150.

a. An artery in transverse section.

d. Ducts of serous glands in transverse section.

l. Lymphatics invaginating the ducts and the artery.

Fig. XXV.

From a similar preparation as the preceding figures. M, p. 200.

d. The duct of a mucous gland, in transverse section.

m. Striped muscular fibres.

l. Lymphatic vessel in connexion with the lymph-spaces around the duct and between the muscular fibres.

Fig. XXVI.

From the same preparation. M. p. about 200.

d. Duct.

l. Lymphatic vessel.

Fig. XXVII.

From the same preparation. M. p. about 150. The lymph-spaces between the muscular fibres are injected with Berlin blue.

t. The bundle of the muscular fibres in transverse section.

l. The same in longitudinal section.

Fig . XXV.

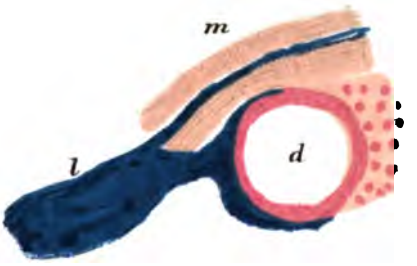


Fig . XXVI.

Fig. XXVII.

Orrcamfield Lith.ᵈ Bedford St Covent Gᵈ

B.

No. 2.

Tabular Statement of Experiments in Inoculation of Bovine Animals with Small-pox at the Brown Institution. By Dr. Klein.

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	Result.
No. 1, heifer, red and white.	1878. March 26	9 punctures	Clear lymph 3rd day*	Pubic region	April 1. The punctures to be felt as small papulæ, three of which show slight redness. April 2. No progress. April 5. All had disappeared.
	April 18	5 pouches† made day previously	Clear lymph 5th day	Side of vulva	No result. All healed on April 21.
	April 22	5 pouches made day previously	Clear lymph 5th day	Side of vulva. The same places as April 22.	April 23. The seat of all inoculations is marked by a papule with central crust. April 25. No progress.
	April 23	5 pouches made day previously	Clear lymph 6th day	Side of vulva	No result.
No. 2, heifer, red and white.	March 26	6 scarifications	Clear lymph 4th day	Pubic region	April 1. Scarifications show no tumidity and no redness. April 4. All are healed.
	April 18	5 pouches made day previously	Clear lymph 5th day	Side of vulva	No result. Healed on April 21.
	April 23	10 pouches made day previously	Clear lymph 6th day	5 on right, 5 on left, side of vulva.	No result.
No. 3, heifer, red and white.	March 26	6 scarifications	Clear lymph 3rd day	Pubic region	April 1. Scarifications show no inflammation, one only is slightly tumid. April 4. All are healing. April 8. All traces lost.
	April 8	5 punctures	Crusts	Side of vulva	April 9. Swelling and redness at seat of inoculation. April 10. Redness diminished, punctures to be felt as tubercles. April 11. Two distinct firm nodular swellings in subcutaneous tissue, corresponding to two punctures. April 17. One opened and found to contain pus.
	April 21	5 pouches made day previously	Clear lymph 5th day	Side of vulva	April 22. Slight tumidity and redness of seat of inoculation. April 23. No progress.

* After the appearance of the eruption.

† *i.e.*, a deep incision (about ¼-inch deep) made with a lancet in a very oblique direction on the day preceding the inoculation. Next day, *i.e.* the day of inoculation, the pouch, which shows only very slight inflammatory change, is opened, and all clots are cleared away with filter paper. The variola lymph is then emptied directly into it from the tube; in this way a greater quantity of lymph than in any other method can be introduced. Generally for each pouch as much lymph as is contained in two medium-sized human variola-pustules is used.

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	Result.
No. 4, heifer, red and white.	1878. March 26	6 scarifications	Clear lymph 3rd day	Pubic region	April 1. No tumidity or redness. April 4. Scarifications are healing. April 8. All healed up.
	April 18	5 pouches made day previously	Clear lymph 5th day	Side of vulva	No result. Healed up on April 21.
	April 22	5 pouches made day previously	Clear lymph 5th day previously.	Side of vulva. Same places as April 18.	April 23. Four very distinct papules with central crust; no redness.
	April 23	5 pouches made day previously	Clear lymph 6th day	Side of vulva	No result.
No. 5, heifer, red and white.	March 26	6 scarifications	Clear lymph 6th day	Pubic region	April 1. Two of the scarifications are slightly tumid. April 4. All except one healing; this one has still its scab, but shows no tumidity or redness. April 8. All healed up.
	April 3	Injection	Clear lymph 5th day	Subcutaneous vein of ear-lobe.	April 4. Skin around wound swollen and red; painful. April 8. Still swollen, but not red or painful. Suppuration of wound. April 10. Healing.
	April 18	5 pouches made day previously	Clear lymph 5th day	Side of vulva	April 23. Seat of inoculation nearly lost, being healed up.
No. 6, heifer, white.	March 26	6 scarifications	Clear lymph	Pubic region	April 1. No redness or tumidity. April 4. Healed.
	April 3	Injection	Clear lymph 5th day	Subcutaneous vein of ear-lobe.	April 4. Skin around wound swollen, red, and painful. April 8. Still swollen, but not red or painful; thick scab covering wound. April 10. Healing.
	April 21	5 pouches made day previously	Clear lymph 5th day	Side of vulva	April 23. Seat of inoculation tumid and slightly red.
No. 7, heifer, red and white.	March 26	12 points	Clear lymph	Pubic region	April 1. No inflammation of any special kind.
	March 27	4 punctures	Clear lymph 3rd day	Pubic region.	April 2. Punctures not to be seen any more. April 4. All traces lost.
	April 9	4 punctures	Clear lymph 5th day	Right side of vulva	April 11. Slight swelling of all punctures. April 13. No progress. April 15. All have disappeared.
		4 punctures	Purulent lymph 6th day	Left side of vulva.	
	April 15	6 punctures	Crusts	Side of vulva	April 17. Punctures recognisable by small scabs.
	April 17	5 punctures	Crusts	Same place as April 15.	April 18. No tumidity or redness. April 21. All traces lost.

TABULAR STATEMENT of EXPERIMENTS in INOCULATION of BOVINE ANIMALS with SMALL-POX—*continued.*

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	Result.
No. 8, heifer, white.	1878. March 26	12 punctures 4 punctures	Clear lymph Clear lymph 5th day	Pubic region Pubic region.	April 1. Punctures to be felt as small papules. The places where points had been used show no change. April 4. Papules still to be felt, but no progress. April 8. All traces lost.
	April 8	8 punctures	Crusts	Side of vulva	April 9. Swelling and redness at seat of inoculations. April 10. Less swelling and redness. April 11. Two red nodules only to be felt and seen. April 13. No progress. April 15. The two nodules may be still felt, but no progress.
	April 15 April 17	5 punctures 5 punctures	Clear lymph 5th day Crusts	Side of vulva Side of vulva.	April 18. Slight tumidity and redness at seat of second inoculation (crusts). April 21. Two or three small pale papules on left side of vulva. April 22. No progress.
No. 9, heifer, red and white.	March 27	10 punctures	Clear lymph 5th day	Pubic region	April 1. Punctures to be felt as small papules, but no redness. April 2. Papules less distinct. April 4. Some of the papules have disappeared. April 8. All have disappeared.
	April 8	6 punctures	Crusts	Pubic region	April 9. Swelling and redness at seat of inoculation. April 10. Less swelling and redness. April 13. All traces lost.
	April 15 April 17	6 punctures 6 punctures	Clear lymph 6th day Crusts	Side of vulva Side of vulva.	April 18. Slight tumidity at seat of second inoculation only. April 21. No trace left.
No. 10, heifer, red and white.	March 27	10 punctures	Clear lymph 5th day	Pubic region	April 1. Punctures to be felt as small papules, three are slightly red. April 4. No progress. April 8. All lost.
	April 9	4 punctures 4 punctures	Clear lymph 5th day Purulent lymph 6th day	Right side of vulva Left side of vulva.	April 11. Punctures slightly tumid on right side only. April 13. All punctures but two disappeared. April 15. The two last disappeared.
	April 15 April 17	6 punctures 4 punctures	Crusts Crusts	Side of vulva Same places as April 15.	April 18. No humidity or redness. April 21. No trace of inoculation.

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	Result.
	1878.				
No. 11, small milch cow.	*a.* June 27	10 deep pouches	Clear fluid lymph	Side of vulva	None, except slight tumidity on the first two days after the inoculation.
	b. July 18	7 compound scarifications	Clear lymph on points and in tubes.	Udder	None; one scarification only surrounded by a red areola, which lasted, however, only the first day after inoculation.
	c. August 7	8 compound scarifications	Clear lymph on points	Udder	None.
No. 12, big milch cow.	*a.* June 27	10 deep pouches 2 valvular incisions 1 compound scarification	Clear fluid lymph " " " "	Side of vulva Udder.	None, except slight tumidity in the first two or three days after the inoculation.
	b. June 28	10 valvular incisions	Clear fluid lymph	Udder	One of the valvular incisions showed a red areola, but only for three days, and then subsided.
	c. July 18	9 compound scarifications	Clear lymph on points and in tubes.	Udder	None.
	d. August 3	10 compound scarifications	Clear lymph on points	Udder	None.
No. 13, big heifer nearly white.	*a.* June 29	7 deep pouches 3 valvular incisions 10 valvular incisions	Clear fluid lymph " " " "	Side of vulva Side of vulva. Udder.	None, except slight tumidity during two or three days after inoculation.
	b. August 2	6 compound scarifications	Clear lymph on points and in tubes.	Udder	None.
No. 14, big heifer nearly white.	*a.* June 30	21 valvular incisions	Clear fluid lymph	Udder	None, except slight tumidity during the first two or three days.
	b. August 1	9 compound scarifications	Clear lymph on points and in tubes.	Udder	None.
	c. August 2	5 compound scarifications	Clear lymph on points and in tubes.	Side of vulva	None.

Tabular Statement of Experiments in Inoculation of Bovine Animals with Small-pox—*continued.*

Animal.*	Date of Inoculation.	Mode of Inoculation.	Material.†	Seat of Inoculation.	Result.
No. 15	1878. August 21	8 compound scarifications‡	Clear lymph (*see note* †)	Udder§	None.
	September 6	6 compound scarifications and 3 valvular incisions.‖	-	Udder	On September 7 there was slight redness round two compound scarifications, but passed off next day.
	September 13	4 compound scarifications and 4 valvular incisions.	-	Udder	None.
No. 16	August 22	6 compound scarifications	-	Udder	None.
	September 6	6 compound scarifications and 3 valvular incisions.	-	Udder	On September 7 slight redness round two or three spots, but passed off after a day.
	September 13	5 compound scarifications and 2 valvular incisions.	-	Udder	None.
No. 17	August 22	7 compound scarifications	-	Udder	None.
	September 7	6 compound scarifications and 3 valvular incisions.	-	Udder	
	September 12	5 compound scarifications and 2 valvular incisions.	-	Udder	
No. 18	September 5	8 compound scarifications and 4 valvular incisions.	-	Udder	On September 6 there was slight redness round one compound scarification, and one valvular incision, but passed off next day.
	September 7	7 compound scarifications and 2 valvular incisions.	-	Udder	None.
	September 12	5 compound scarifications and 2 valvular incisions.	-	Udder	On September 13 one spot much swollen. On September 15 all right again.

* All 4 animals were *heifers, mostly white.*

† In all cases *clear lymph* of 4th or 5th day was used: it was derived from *confluent variola* of *unvaccinated* individuals, and was used fluid in tubes, and dry on points.

‡ Each *compound scarification* consists of 10 *or* 12 *incisions* in one direction, and 10 *or* 12 *incisions crossing the first;* they cover an area of $\frac{1}{4}$ *or* $\frac{1}{2}$ *square inch.*

§ The back part of udder and adjoining pubic region being used.

‖ Each *valvular incision* is about $\frac{3}{4}$ *of an inch long,* and about $\frac{1}{4}$ *of an inch broad.*

Each *compound scarification* received *three large points,* charged three and four times on both sides; in addition to this each compound scarification was *flooded with fluid lymph.* Each *valvular incision* received *one large point,* and in addition was *flooded with fluid lymph.*

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	Result.
No. 19,* white milch cow (had calf).	1878. *a.* October 24 - *b.* November 12 *c.* December 11	*a.* 8 compound scarifications and 3 simple scarifications. *b.* 5 compound scarifications and 5 simple scarifications. *c.* 5 compound scarifications and 5 simple scarifications.	Clear lymph of confl. var. on points and fluid.	Udder - - -	The inoculated surface of udder of these three cows was covered and kept warm with a cloth for the first week after each inoculation. No result.
No. 20,* red and white milch cow (had calf).	*a.* October 24 - *b.* November 18 *c.* December 11	*a.* 6 compound scarifications and 4 simple scarifications. *b.* 6 compound scarifications and 4 simple scarifications. *c.* 5 compound scarifications and 5 simple scarifications.	Clear lymph of confl. var. on points and in tubes.	Udder - - -	
No. 21,* white milch cow (in calf).	*a.* October 25 - *b.* November 6 *c.* November 12	*a.* 8 compound scarifications and 5 simple scarifications. *b.* 5 compound scarifications and 5 simple scarifications. *c.* 5 compound scarifications and 4 simple scarifications.	Clear lymph of confl. var. on points and in tubes.	Udder - - -	
No. 22,* white milch cow (had calf).	1879. *a.* January 16 *b.* January 30 *c.* February 6	*a.* 6 compound scarifications and 4 simple scarifications. *b.* 5 compound scarifications and 4 simple scarifications. *c.* 3 compound scarifications and 3 simple scarifications.	Clear lymph of confl. var. on points and in tubes.	Udder - - -	The inoculations were covered with hot poultice which was renewed every day for several days. No result.
No. 23,* white milch cow (in calf).	*a.* January 16 *b.* January 30 *c.* February 6	*a.* 6 compound scarifications and 4 simple scarifications. *b.* 6 compound scarifications and 3 simple scarifications. *c.* 3 compound scarifications and 3 simple scarifications.	Clear lymph of confl. var. on points and in tubes.	Udder - - -	

* *See* Note * on page 142.

TABULAR STATEMENT of EXPERIMENTS in INOCULATION of BOVINE ANIMALS with SMALL-POX—*continued.*

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	RESULT.
No. 24,* white and grey milch cow (in calf), Alderney.	1879. *a.* March 13 *b.* April 16	*a.* 5 compound scarifications and 6 simple scarifications. *b.* 5 compound scarifications.	Clear lymph of confl. var. on points and in tubes.	Udder	Even after one month two compound and one single scarification were not healed, but were still covered with crust, which when removed left a slight soreness behind.
No. 25,* Alderney milch cow, red and white (in calf).	*a.* March 13 *b.* April 16	*a.* 5 compound scarifications and 5 simple scarifications. *b.* 5 compound scarifications.	Clear lymph of confl. var. on points and in tubes.	Udder	After one month one place still covered with scab; when removed slight soreness.
No. 26,* red and white milch cow in calf.	*a.* May 7 *b.* May 21 *c.* June 13	6 compound scarifications and 4 valvular incisions. 4 compound scarifications and 4 valvular incisions. 8 valvular incisions.	Clear lymph of confl. var. on points and in tubes.	Udder	No result.
No. 27,* white milch cow in calf.	*a.* May 7 *b.* May 21 *c.* June 13	6 compound scarifications and 4 valvular incisions. 4 compound scarifications and 4 valvular incisions. 8 valvular incisions.	Clear lymph of confl. var. on points and in tubes.	Udder	No result.
No. 28,* white milch cow in calf.	*a.* May 7 *b.* May 21 *c.* June 13	6 compound scarifications and 4 valvular incisions. 4 compound scarifications and 4 valvular incisions. 8 valvular incisions.	Clear lymph of confl. var. on points and in tubes.	Udder	No result.
No. 29,* white and red milch cow in calf.	*a.* May 15 *b.* June 6 *c.* June 18	5 compound scarifications and 5 valvular incisions. 9 valvular incisions. 8 valvular incisions.	Clear lymph of confl. var. on points and in tubes.	Udder	No result.

* *See* Note * on page 142.

App. B. No. 2.

On Inoculation of Bovine Animals with Small-pox, by Dr. Klein.

Animal.	Date of Inoculation.	Mode of Inoculation.	Material.	Seat of Inoculation.	Result.
No. 30,* red and white milch cow in calf.	1879. a. May 15 -	5 compound scarifications and 5 valvular incisions.	Clear lymph of conf. var. on points and in tubes.	Udder - - -	None.
	b. June 6 -	9 valvular incisions.			
	c. June 13 -	8 valvular incisions.			
No. 31,* white milch cow in calf.	a. May 15 -	5 compound scarifications and 5 valvular incisions.	Clear lymph of conf. var. on points and in tubes.	Udder - - -	None.
	b. June 6 -	9 valvular incisions.			
	c. June 13 -	8 valvular incisions.			

* In these 13 animals, except Nos. 24 and 25, there was no sign of redness or inflammation. The places of inoculation were slightly tumid a day or two after the experiment, but became less so in a short time. They were covered with brown scabs, which could be easily removed after seven or eight days, and fell off spontaneously after about 10 days. At no time were they other than dry, and the place of inoculation was likewise at all times perfectly dry and lymphless. In Nos. 24 and 25 (Alderney cows) some of the places of inoculation took a very long time to heal, but were always lymphless and only slightly tumid in the first few days.

NOTE.

As bearing on the question whether the inoculation of bovine animals with human variola, lymph produces any constitutional effect, it may be useful to state that several of the above animals were soon after the variolous inoculation described in the table, subjected to inoculation with humanized vaccine lymph; and that they appeared insusceptible to it in proportion to the amount of previous "variolation." In two cases (heifers No. 5 and 6) the "vaccination" with fresh and good humanized vaccine lymph took place after the animals had been three times variolated in two others (milch cows, Nos. 22 and 23) after the animals had been twice variolated, and in two further instances (milch cows Nos. 24 and 25) the "vaccination" was performed simultaneously with the second variolation. It was found that in the first three animals (No. 5, No. 6, and No. 22) no effect whatever was produced by the vaccination; in No. 23, only a slight result was obtained, viz., out of 6 scarifications, inoculated with humanized vaccinia, one led to ulceration, without passing, however, through the stage of the vesicle. In each of the last two cows (24 and 25) five days after the operation, all (four) places of vaccination were swollen, red, and indurated; they did not lead to vesicles, but passed into small sores.

B.

No. 3.

Further Researches on the Chemical Constitution of the Brain, and of the Organoplastic Substances, by J. L. W. Thudichum, M.D., F.R.C.P.

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II.—Further Researches on the Chemical Constitution of the Organoplastic Substances.

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I.—Separation of the Nitrogenised Principles of the Brain.

A. *Spirit Treatment.*—A quantity of white matter, from which lecithin and kephalin had been removed by treatment with ether, and which had been recrystallised from spirit eight distinct times, was selected for purification. The mixture, containing phrenosin, kerasin, cerebrinic acid, and myelin, was brought to the consistence of a cream by rubbing in a mortar with spirit of 85 °/₀, and was added in small quantities at a time to hot spirit in a platinum vessel. This was done in order to effect as perfect a solution as possible of the soluble part, and to prevent as far as possible the formation of the insoluble matter termed "stearoconote." (See Reports, New Series, No. III. (1874), p. 210.) The solution was filtered hot and set aside to deposit. On cooling a white body came down and was collected. On analysis this was found to contain 0·856 per cent. of phosphorus. This process was repeated a tenth time, and the product, having lost but little of the phosphorised

ingredient (myelin, which in spirit of 85 °/₀ strength has the same solubility as the cerebrin bodies) was subjected to the treatment with plumbic acetate described on p. 187 of the report already referred to.

B. *Lead Acetate Treatment.*—The body was triturated with *alcoholic* solution of lead acetate, and the mixture poured as before into hot spirit: the solution was filtered hot, and allowed to cool. The deposit was collected, and treated with more alcoholic solution of lead acetate, and filtered from the excess of liquid. After this it was twice dissolved in hot spirit to remove excess of lead acetate. The deposit, on analysis, was found to contain 0·73 °/₀ of phosphorus.

The body was now triturated with watery solution of lead acetate, and pressed to remove the excess of liquid. The mass was made into a cream with cold spirit, and added in small quantities to hot spirit. The solution was filtered and set aside to cool, and the stearoconotised portion was put aside for separate treatment. The alcoholic and watery filtrates from which all matters deposited on cooling had been removed were concentrated, and treated as will be described elsewhere. The white body which came down from the spirit solution on cooling was collected and treated again with spirit, and this process was repeated until no more so-called stearoconote (or lead precipitate) was produced. The ultimate deposit obtained, after all these recrystallisations had been carried out, was collected and dried at 100° C. At this temperature it did not alter in appearance. It gave a purple reaction with oil of vitriol alone, and on analysis was found to contain 0·73 °/₀ of phosphorus.

C. *Absolute Alcohol Treatment without Fractionation of Precipitate.*—Absolute alcohol was now used in place of spirit of 85 °/₀, and the solution and recrystallisation were repeated a great number of times until the the mother-liquors gave no more precipitate with cadmic chloride. As long as the alcoholic filtrate gave a precipitate with this reagent, it was manifest that it removed the phosphorised myelin, and as will be seen below, the separation of the phosphorised part succeeded, gradually but effectually, to the extent of concentrating the phosphorus in the part soluble in the cold, so that it contained nearly 2 per cent. (1·951 °/₀), while the part insoluble in the cold retained only one tenth of that amount. But when the solubility of the phosphorised ingredient in absolute alcohol had become again equal to that of the nitrogenised substance, it was found requisite to resort to fractional precipitation for the purpose of isolating pure educts.

D. *Separation of Phrenosin and Kerasin by Fractional Precipitation on Cooling.*—It had been observed that when the absolute alcohol solution began to deposit matter, which occurred between 50° and 40°, rosettes of phrenosin appeared first. When the temperature reached 28° this ceased, and the supernatant liquid was clear for a while, until the temperature fell to 26°. Below this temperature a gelatinous cloudy mass, mainly of kerasin, gradually formed and floated on the phrenosin.

The phrenosin was therefore isolated as follows. When the temperature of the liquid in which the phrenosin crystallised had fallen to 28°, the mother-liquor which contained most of the kerasin in solution was swiftly decanted, either with or without the employment of a filter kept at 28° by a water-bath; and the phrenosin was thus recrystallised seven times until no further separation seemed to be effected.

An analysis of the phrenosin at this stage gave phosphorus equal to 0·182 °/₀, and after two further treatments in the same way it yielded phosphorus equal to 0·113 °/₀. It also gave in two estimations the following quantities of inorganic matter:—(i) = 0·19 °/₀; (ii) = 0·23 °/₀, containing ·07 of potash (K_2O). The dried kerasin yielded phosphorus

=0·198 °/₀ and potash = 0·07, from which it will be seen that the many resolutions and the several lead treatments had not yet removed all inorganic ingredients from these matters.

E. *Further Purification of Phrenosin by Cadmic Chloride, Ether, and Hydrothion.*—The phrenosin isolated by fractional precipitation as described above was mixed with solution of cadmic chloride, and the mixture suspended in ether. Hydrothion was passed into this, when a yellow solution and a yellow precipitate formed, which remained mixed with the bulk of the undissolved phrenosin. The solid matter was filtered from the liquid; it consisted of phrenosin and cadmic sulphide, while a peculiar compound of myelin with cadmic sulphide (first described in these Reports, New Series, No. III. (1874), p. 169), remained in solution. The residue was removed from the filter and dissolved in hot 85 °/₀ spirit. The insoluble cadmic sulphide was filtered off, and the solution was set aside to deposit. On cooling phrenosin came down in large rosettes, and was collected and recrystallised, first twice from 85 °/₀ spirit, and finally from absolute alcohol. It was dried and then exhibited the following properties. When boiled with water it no longer swelled to a starchy paste, but merely became flocculent and floated about in the fluid. Treated by itself in the cold with oil of vitriol it very slowly developed a purple reaction, but more quickly when warmed. It passed through an intermediate yellow stage—particularly well marked in the reaction—which is obtained without the employment of heat, and during which the matter is wholly in solution. Then flocks separate and slowly become purple.

Two estimations of phosphorus were made: No. 1 gave 0·045 °/₀, and No. 2 gave 0.051 °/₀. Taking the mean of these two results, we have 0·048 °/₀ of phosphorus. Calculated as myelin, this would give 1 °/₀ as the amount of phosphorised substance left in the phrenosin.

At this stage of its purification the phrenosin was subjected to elementary analysis.

II.—Elementary Analysis of Phrenosin, $C_{41}H_{79}NO_8$.

A. *By Vacuum Method.*—The substance was burnt with oxide of copper and copper *in vacuo*, the resulting water was weighed, and the gaseous mixture of carbonic acid and nitrogen was analysed and estimated volumetrically.

(*a.*) 0·0994 grm. of the substance previously dried over oil of vitriol *in vacuo* gave on combustion 0·1050 grm. H_2O, 0·2468 grm. CO_2, and 0·00214 grm. N.

The data from which the two last numbers are calculated are given in the following table:—

—	Volume.	Temperature.	Pressure corrected for aq. vap. etc. and reduced to 0° C.
Gas before potash treatment.	156·80 c.c. moist.	15°·1 C.	648·22 m.m.
Gas after potash treatment..	6·84 c.c. moist.	15°·2 C.	199·73 m.m.

These figures correspond to 67·71 per cent. carbon, 11·63 per cent. hydrogen, and 2·15 per cent. nitrogen.

(*b.*) 0·1113 grm. dry *in vacuo*, burnt, gave 0·1144 grm. H_2O, 0·2771 grm. CO_2, and 0·00237 grm. N.

The data from which the two last numbers are calculated are contained in this table:—

—	Volume.	Temperature.	Pressure corrected for aq. vap. etc. and reduced to 0° C.
Gas before potash treatment.	167·19 c.c. moist.	16°·7 C.	677·23 m.m.
Gas after potash treatment.	8·295 c.c. moist.	15°·7	182·6 m.m.

These numbers lead to 67·89 per cent. carbon, 11·42 per cent. hydrogen, and 2·13 per cent. nitrogen.

(*c.*) 0·1079 burnt, gave 0·1091 grm. H_2O, 0·2677 grm. CO_2, and 0·00223 grm. N.

The data for calculating the latter two figures are shown in the following table:—

—	Volume.	Temperature.	Pressure corrected for aq. vap. etc. and reduced to 0° C.
Gas before potash treatment.	161·80 c.c. moist.	10°·2	668·7 m.m.
Gas after potash treatment.	7·472 c.c. moist.	10°·1	187·04 m.m.

These data lead to 67·67 per cent. of carbon, 11·23 per cent. of hydrogen, and 2·07 per cent. of nitrogen.

B. *By Liebig's and by Dumas's Method.*—(*d.*) 0·3350 grm. burnt with copper oxide and copper according to Liebig's method gave 0·8424 grm. of CO_2, equivalent to 68·56 per cent. of carbon.

(*e.*) 0·5230 burnt with copper oxide and copper in an atmosphere of carbonic acid according to the method of Dumas, as modified by Thudichum and Wanklyn, gave 13·2 c.c. nitrogen gas; temp. 20° C; bar. 757·3 m.m.; difference of Hg levels 134 m.m.; height of potash column 161 m.m., therefore V = 0·01198 grm. N, equivalent to 2·29 per cent.

(*f.*) 0·7025 grm. burnt as in previous analysis gave 13·7 c.c. nitrogen gas; temp. 16° C; bar. 752 m.m.; mercury level, potash column 220 m.m.; therefore V = 0·01531 grm. N, equivalent to 2·18 per cent.

(*g.*) 0·6625 grm. was burnt in CO_2 with CaO and Ca, and gave 12·406 c.c. of gas, normal, equivalent to 2·34 per cent. N.

C. *By Will and Varrentrapp's Method.*—(*h.*) 1·154 grm., burnt with soda lime, gave 0·296 grm. of Pt $Am_2 Cl_6$, and 0·013 grm. Pt, equivalent to 1·768 per cent. N.

(*i.*) 1·2725 grm. burnt with soda lime yielded 0·327 grm. Pt $Am_2 Cl_6$, and 0·007 grm. Pt, equivalent to 1·69 per cent. N.

(*k.*) 0·940 grm., burnt with soda lime, gave 0·114 grm. Pt, equal to 1·715 per cent. N.

The per-centages obtained in the foregoing analyses are compared in the following table:—

—	*a.*	*b.*	*c.*	*d.*	*e.*	*f.*	*g.*	*h.*	*i.*	*k.*
C	67·71	67·89	67·67	68·56	—	—	—	—	—	—
H	11·63	11·42	11·23	—	—	—	—	—	—	—
N	2·15	2·13	2·07	—	2·29	2·18	2·34	1·768	1·690	1·715
O	18·51	18·56	19·03	—	—	—	—	—	—	—
—	100·00	100·00	100·00	—	—	—	—	—	—	—
—	By combustion in vacuo; C and N volumetrically; H_2O weighed.			CO_2 weighed.	By combustion in CO_2 atmosphere; gas volumetrically estimated.			By combustion with soda-lime; Pt. salt weighed.		

D. *Consideration of the Methods of Analysis.*—The quantation of the nitrogen as gas, whether it has been obtained by combustion in a vacuum, or by combustion under atmospheric pressure in an atmosphere of carbonic anhydride, always gives the nitrogen a little too high as compared with theory, whereas the estimation of nitrogen by transformation into ammonia always gives the amount of this element a little lower than is required by theory. These discrepancies are well known to be inherent in the methods. They are less significant as regards the analysis of bodies which are rich in nitrogen, than in the analysis of bodies containing only a small per-centage of this element. In the particular case of phrenosin they are so great as to make it at first sight impossible to derive a correct empirical formula from the data given by either mode of analysis. But *the mean* of the data for nitrogen obtained by the three different modes of analysis is very nearly coincident with the sum of the data obtained by the chemolytic method. This coincidence is probably the result of accident only, at least there is at present no explanation derivable from the most searching scrutiny of either the methods employed, or of the particular manner in which they have been executed.

In the vacuum analysis the carbon is regularly found a little too low; yet not a trace of carbon is left unburned in the tubes, as was specially proved; moreover the combustion tubes with their contents were weighed, before and after combustion, and the weights of the sums of the products of combustion, as calculated from their volumes in the case of carbon and nitrogen, showed an almost mathematical coincidence with the respective losses which the tubes had undergone.

In this case of phrenosin the known methods of elementary analysis are therefore unavailing to lead to final results; on the contrary, phrenosin is a good test object by the use of which the particular failings as well as strong features of these methods can be made apparent. Thus, combustion, according to Liebig, of not too small a quantity of phrenosin, yields the best carbon estimate, combustion in vacuo of such a small quantity as that to which the method is necessarily limited, the worst.

The very same methods which in the case of phrenosin yield the results discussed in the foregoing, give, when applied to its decomposition products, results which are in much greater concordance with the requirements of theory. This unquestionable fact shows that the size of the molecle, and the proportions in which the elements contained stand

to each other, have an influence on the result of the process of elementary analysis; a simple compound of fewer atoms gives more accurate results when tested by different methods, while a more complicated compound with many atoms, when tested in the same manner by different methods, give much less accurate results. We may, therefore, expect that the elementary analysis of the more complicated nitrogenised bodies, such as kerasin and cerebrinic acid, will present even greater difficulties than those which have been experienced in the analysis of phrenosin.

III.—Chemolysis of the Nitrogenised Principles of the Brain by Sulphuric Acid in Watery Solution.

A. *Introduction.*—In these Reports, New Series, No. VIII. (1876), p. 138, I have described some experiments intended to throw further light on the constitution of the nitrogenised principles of the brain as defined on p. 182 of No. III. of the New Series of these Reports. A sugar was then isolated in an uncrsytallisable state and subjected to elementary analysis. But the other products of the reaction were not sufficiently defined, partly from want of material, partly from want of apparatus of sufficient capacity to effect a reaction within a reasonable limit of time. In my earliest chemolyses with baryta, hermetically sealed glass tubes, enclosed in iron tubes were employed. (See p. 134 of Report, New Series, No. VIII.) But of these at least half succumbed to the internal pressure, and their contents were lost. I therefore procured a special tube of brass lined with platinum, described on p. 137 of Report VIII. This worked satisfactorily for the baryta chemolyses which were effected in a short time; but the sulphuric acid chemolyses were found for their completion to require the influence of a temperature of at least 130° for a period varying from 310 to 370 hours. It was therefore necessary to multiply the tubes, and in order to do this consistently with practical considerations leaden tubes were employed. Such tubes could not have been employed in the baryta chemolysis, as baryta rapidly and energetically attacks lead. But dilute sulphuric acid has but a slight influence on metallic lead, and the small quantity of metal which is dissolved is easily removed from the organic products by appropriate means.

B. *The Apparatus.—The Leaden Tubes.—The Hot-Air Stove.*—The leaden tubes were an inch in calibre; the metal was an eighth of an inch thick; each tube was eighteen inches long. One end of the tube was closed by hammering only so as to form a semi-globular end, not larger in diameter than the tube itself; it was tested by water, and when none passed it was soldered over on the outside. The mixture to be chemolysed was now put into the tube by means of a wide tubed funnel; the upper part of the tube was heated to dry it completely and rarefy the air in the air-space, which amounted to about one-sixth of the length of the tube. The mouth of the tube was now suddenly compressed in a vice and closed; the edges of the lead were filed smooth, moistened with zinc chloride, and immediately soldered, or as the operation is technically termed "burnt," with the oxyhydrogen flame.

The hot-air stove was made of copper, and consisted of two horizontal air cushions, between which special room for six of the leaden tubes just described was arranged so that their longest axes were lying in a horizontal position. The tubes could thus be kept at an equable temperature, which could be read by the thermometer, the ball of which was in the central air-space, while the stem projected over the top of the stove. The stove was heated by aërated gas flames.

C. *Preliminary Purification of the Nitrogenised Substances to be chemolysed.*—As these substances retain inorganic salts with great pertinacity they were subjected to a process for the removal of these. They were boiled in water until completely disintegrated, and the starch-like solution was pressed through a cloth. The filtrate was then mixed with sulphuric acid sufficient for it to contain one per cent. and boiled for one hour. The nitrogenised matters are soon curdled out of the solution, free from salts but partially altered; for the acid solution after treatment with baryta carbonate is found to contain considerable quantities of chemolytic sugar (cerebrose) besides some alkaloidal matters, and the potassium, sodium, and earthy salts which it was the object of the process to remove.

D. *Chemolysis of the Purified Substance.*—Six leaden tubes, prepared as above, each received about 30 grms. of nitrogenised substance purified as described, and 353 c.c. of dilute sulphuric acid containing 2 % H_2SO_4. The tubes were then closed as described, placed in the hot-air stove, and heated to 130° during 24 hours. After the lapse of this period the tubes were opened at the compressed end with a chisel, the dilute acid was removed and filtered, all solid matter was kept in the tubes or returned to them together with a fresh charge of acid. The tubes were again closed as described and heated for a second 24 hours. This treatment was repeated as long as the acid liquid contained any cerebrose, and the chemolyses were deemed complete only when the last charge of dilute acid was found on proper treatment and concentration to be free from cerebrose. This result was in most cases not attained in less than 14 days, in some required from 16 to 17 days, in a few even 24 days, during which the chemolysis was continued day and night.

E. *The Acid Filtrates.*—These were boiled with barium carbonate, prepared pure by precipitation for the purpose. The neutral filtrates were evaporated to about one-fifth of their bulk in a water bath, then removed into a distilling apparatus connected with an air-pump, and distilled at a temperature of from 30° to 40° to the consistency of a syrup. The latter was put aside to crystallise. Stellate groups of crystals soon formed, and the entire syrup gradually solidified to a granular mass of crystals. These were separated partly by drawing off the small quantity of mother-liquor, partly by agitating them with water, and getting rid of the mother-liquor by dilution; for the crystals were but slowly soluble in cold water.

From the mother-liquor a further quantity of crystals was obtained by addition of boiling alcohol to the hot concentrated aqueous solution, until a considerable permanent turbidity was produced. When the mixture was allowed to stand in the cold for one day, it formed a considerable amount of a coloured deposit, from which the clear supernatant alcoholic liquid was decanted. This latter solution on standing for some weeks, and after repeated additions of small quantities of alcohol, and lastly of ether, deposited a crop of white crystals which were added to those obtained in the first operation.

The mother-liquors were, however, always considerable in amount, and after concentration yielded an uncrystallisable syrup, which amounted to about the same weight as the crystals obtained from it. The syrup could be made to crystallise a third time after the removal of some traces of potassium and of a compound ammonium base by platinic chloride, and the removal of all traces of the reagent by hydrothion and silver carbonate.

IV.—Cerebrose, a New Crystallised Sugar.

A. *The Crystals.—Cerebrose* $C_6H_{12}O_6$.—The crystals obtained as described in the foregoing were dissolved in water, and the solution was evaporated in vacuo, after treatment with animal charcoal. A mass of crystals was again obtained, which were smaller than the first ones, but perfectly white and very hard. The colourless mother-liquor of these on standing over oil of vitriol solidified to a hard mass of crystals, which rose much over the level of the liquid in which they formed. The crystals were not large enough for crystallometric treatment; seen under the microscope they seemed to consist of rhombic octahedra, of which some were elongated to prisms. This body and the accompanying syrup are the same as that of which a preliminary description has already been given on p. 140 of these Reports, New Series, No. VIII. As it is now fully shown to be a kind of sugar hitherto unknown, I shall signalise it hereafter by the name of "*Cerebrose.*" It was again subjected to elementary analysis, and gave analytical data, leading to formula $C_6H_{12}O_6$.

B. *Reducing Power of Cerebrose over Cupro-Potassic Tartrate.*—Cerebrose reduces Fehling's solution readily on heating, and the precipitated suboxide of copper is mostly of a dark red colour. 0·2622 grms. cerebrose dried in the water-bath were dissolved in water, and the solution made to fill the space of 50 c.c. This solution, therefore, contained 0·5245 % of cerebrose. It was employed to reduce a Fehling's solution of which 5 c.c. required 0·025 grms. of glucose for decoloration. 5 c.c. of the Fehling's solution required 5·6 c.c. of the cerebrose solution for decoloration, equal to 0·0294 grms. cerebrose. A certain quantity of cupro-potassic tartrate, therefore, which requires five parts of glucose for complete reduction, requires about six parts of cerebrose for reduction.

C. *Polarising Power of Solution of Cerebrose.*—Some cerebrose was dried in the air-bath at 90°, and in vacuo till it remained constant in weight. Of this 2·6315 grm. were dissolved in water with the aid of a gentle heat, and made to fill a space of 20 c.c. The solution was treated with animal charcoal to remove a slight turbidity. Its strength remained at 13·16 %. This in a tube of 100 m.m. length rotated the ray of polarised light at 25° T = +10° 40′. But after 24 hours standing the same tube at T 11° rotated only +9° 24′; after a second 24 hours at T 12° = +9° 18′. The solution was then diluted with an equal bulk of water, and its rotation, measured in a tube twice the length of the former, was found to be +9° 24′ at 9° T to +9° 32′ at 8° T. These data by means of the usual calculation lead to the specific or limited rotation for cerebrose of +70° 40′. It will be observed that immediately after solution the rotation is a little higher than 24 hours after solution, when it becomes constant. But this increase of the rotation is very slight compared with the increase which dextro-glucose exhibits immediately after solution, and which, from the fact of its being about as much again as the constant rotation, is termed the birotation of glucose.

In Hermann's Physiology, there occurs a statement that a sugar obtained from the so-called Protagon was lævo-rotatory. From the foregoing it will be evident that the author never isolated the sugar yielded by the nitrogenous substances of the brain, which is dextro-rotatory. This sugar cerebrose was for the first time isolated in the researches here reported.

The actual molecular size of cerebrose, assumed in the foregoing to be $C_6H_{12}O_6$, cannot at present be given with certainty. It might be a multiple of this formula, or at least its double, and thus belong to the

thickened, apparently from swelling of bundles of fibrous tissue, among which, in some parts, was an unusually large number of connective-tissue corpuscles. No micrococci were found.

Suprarenal capsule.—Some capillaries filled with micrococci. No other change observed.

Kidney.—In some sections was to be seen very clearly what appears to be the normal relation of the basement membranes to the epithelium of the collecting tubules, but which has not, so far as I can ascertain, been previously described. The membrane sends in processes between the epithelial cells so as when cut vertically to show when the loosened epithelium has fallen out, a number of projecting points and a crenated border towards the lumen of the tube. When the tubule is seen from the surface these processes appear as lines running transversely to the axis of the tube, and when the epithelium has fallen out, resemble the rungs of a ladder. When the section has cut the tubule obliquely they resemble re-duplications of the inner part of the membrane like valves in a vein. Plate IX., Fig. 2, shows the arrangement very clearly, but they may be possibly due to some commencing pathological change. Since, however, my attention was directed to it by this specimen, I have observed more or less distinctly a similar arrangement in many other sections from different kidneys. In the basement membrane are also to be seen stellate cells. In other sections from this kidney the epithelium of the convoluted tubules was so swollen as to altogether obliterate the lumen of the tubes. The nuclei were increased in number, and these with the compressed capillaries, the endothelial nuclei of which were more distinct than usual, alone served to distinguish the tubules from each other. The glomeruli were in many parts with greatly increased numbers of nuclei, and the larger vessels were much thickened. More particularly the middle coat, which in some places had lost all trace of any structure, and presented a homogeneous appearance deeply stained with hæmatoxylin.

Liver.—Some capillaries filled with micrococci. Liver-cells swollen, cloudy, and abundantly infiltrated with fat. Many also contained an abundance of bright yellow pigment granules. There were many connective-tissue corpuscles in the sustentacular structure, and colourless corpuscles in the lymph spaces between the liver-cells and blood-vessels.

Spermatic cord.—The connective tissue of the cord was crowded with small, round cells, and in almost every section examined were miliary abscesses.

Case 21.* *Acute necrosis of humerus; phlebitis of brachial veins; secondary abscesses in lungs, heart, spleen, and kidneys.* Organs examined—Heart, lung, kidney, liver, spleen, lymphatic gland, thrombosed vein.

Heart.—Pericardium much thickened in parts from swelling of fibrous tissue. Subpericardial vessels distended, some with disintegrating coloured corpuscles, others with fibrinous coagula containing a great number of colourless corpuscles. Adventitia of blood-vessels much hypertrophied. Lymph spaces between muscular fibres of heart and capillaries in many places crowded with colourless corpuscles. In other parts great increase of connective corpuscles. Some capillaries plugged with micrococci. These were found as well in parts of the heart, which microscopically showed no other change; as also in parts which were much altered from the normal. Plate VIII., Fig. 3, shows such a group of micrococci which have partly escaped from the vessel into surrounding tissue, which appears to be normal. The muscle fibre was in some places quite normal, in others marked by double transverse bars with a paler interspace, resembling an exaggerated diagram of the junction of the muscle cells of the heart. Closer examination, however, showed that this appearance resulted from a rupture of the superficial part of the muscular fibre, with contraction and swelling of the ruptured parts, and the remaining portion of the fibre appearing as the paler interspace. Many gradations of this change could be observed in different parts of the same sections. In some fibres the interspace could scarcely be made out, in others the ruptured portions had separated very widely, and the tissue between was much diminished in thickness; in others, the rupture of the fibre was complete, and only irregularly contracted, distinctly separated masses of muscle fibre were to be seen, which in other parts were represented by granular masses showing no trace whatever of

muscle structure. (Plate VIII., Figs. 1 and 2). The heart contained numerous miliary abscesses.

Lung.—Extravasated blood beneath pleura and into lung substance. In some places the blood had escaped through the ruptured pleura on to the surface of the lung. A few groups of capillaries on air-vessels plugged with micrococci. Catarrhal pneumonia.

Kidney.—Epithelium in many parts swollen and pigmented, often completely detached from the basement membrane, which was thicker than normal. Endothelium of blood-vessels much detached. Adventitia of larger vessels swollen. Greatly increased number of nuclei in some glomeruli and afferent vessels thereof. Other glomeruli contracted to one third their normal size. Some capillaries among the convoluted tubules plugged with micrococci. Extravasations of blood beneath capsule.

Liver.—Liver-cells cloudy, granular, almost all studded with bright yellow pigment granules. Intralobular veins thickened, and liver-cells round them shrivelled and in many parts fallen out. Connective tissue of portal canals hypertrophied, and with increased number of cells. Many capillaries in acini plugged with micrococci without any noteworthy difference in liver-cells surrounding them. Some of the larger veins with remains of coagula adhering to the intima, and in them plumose crystals (? tyrosin).

Spleen.—A few capillaries contained micrococci. In many parts the Malpighian corpuscles and splenic pulp cloudy from granular matter between the lymph corpuscles. In other parts the adenoid structure replaced by masses of swollen fibrous tissue most markedly in neighbourhood of larger vessels.

Lymphatic gland.—Much congested; at periphery some extravasation of blood with gland substance. Vessels all distended with coagulated blood. In centre of gland a large vessel cut transversely, showing a smaller lateral branch plugged with micrococci, which projected in a mass with a free rounded extremity into the lumen of the larger vessel.

Thrombosed vein.—Nothing noteworthy.

Case 22. — A. M—. Organs examined — Heart, kidney, liver, spleen, inguinal and lumbar lymphatic glands, and brain.

Heart.—Some muscular fibres, with an early stage of rupture similar to that described in Case 21. Nuclei of muscle fibre very large, and with Klein's network very distinctly visible. Congestion of subpericardial vessels with some small extravasations of blood. No micrococci found.

Kidney.—Great congestion of surface vessels. Glomeruli with increased number of nuclei. Epithelium swollen and cloudy. Some mulberry concretions in collecting tubules. No micrococci found.

Liver.—Hypertrophy of connective tissue round intralobular veins. Cell infiltration of portal sheaths. Liver-cells in many places infiltrated with fat, in others shrivelled, and where they had fallen out thickening of sustentacular structure: some few capillaries plugged with micrococci.

Spleen.—Increased adenoid growth round vessels with commencing granular disintegration of adenoid structure.

Lymphatic glands.—Vessels in capsule with walls much swollen, in some endothelium extensively detached. In the substance of the gland the adventitia much swollen. In many parts gland tissue replaced by considerable quantity of bundles of swollen fibrous tissue, staining pale violet with hæmatoxylin. Between many of such bundles clefts lined with endothelium, suggesting that the change has resulted from hypertrophy and swelling of adventitia of vessels, encroaching upon and replacing gland tissue. In other parts the adenoid structure swollen and similarly stained, with but few lymph corpuscles remaining. In many of the masses of fibrous tissue are giant cells of varying form. In many, however, the nuclei are arranged with such regularity at the periphery as to irresistably suggest their origin from the intima of a blood-vessel. A great many capillaries throughout the gland were plugged with micrococci. A special point of interest in this case is that while no micrococci could be discovered in any of the other organs, every section of the glands sent for examination teemed with these organisms. Among masses of degenerating fibrous tissue many laminated bodies, sometimes irregularly spherical, and frequently trefoil-shaped (Hassel's corpuscles) were found.

Brain.—Numerous corpora amylacea, sometimes isolated, sometimes in groups.

Lungs.—Every section showed numerous vessels, capillaries, and others filled with micrococci. Many bronchial tubes stopped by epithelium and filled with pus. Air-cells contained a very great number of large round cells filled with black pigment. Catarrhal pneumonia in many parts and extravasations of blood.

Liver.—Portal sheaths infiltrated with round cells, which, in many parts, were aggregated in dense round masses. Similar infiltration in centre of many acini. In such acini many liver-cells much chiselled and pigmented. Other parts of section quite normal.

Spleen.—Hyaline degeneration of middle coat of vessels. Great increase in adenoid growth round such vessels. At other parts adenoid tissue degenerating and becoming replaced by swollen fibrous tissue.

Kidney.—Some vessels with micrococci. Great increase of nuclei in glomeruli in afferent vessels. Cellular infiltration of intertubular substance. Extravasations of blood.

Coagula from large veins.—Some sections showed intermingled micrococci.

No other organs sent for examination.

CASE 23. *Thyroid gland.*—To the naked eye this organ appeared so healthy and well preserved that sections were made for demonstration of the normal structure of the gland. Many capillaries were found plugged with micrococci, and in larger vessels masses of these organisms, usually spherical in form, were to be seen in coagula of blood and fibrin. No other change observed.

Lungs—Many of the smaller bronchi stripped of epithelium and filled with pus; catarrhal pneumonia; numerous capillaries on air-cells filled with micrococci.

Heart.—No observed change.

Kidney.—Every section examined contained many groups of micrococci; sometimes in capillaries, in which they could be traced through several branches, sometimes as large round masses, which had escaped from the ruptured vessel. Epithelium well preserved, but in some parts detached from basement membrane. In some tubes mulberry concretions.

Liver.—Some capillaries contained micrococci. Liver-cells infiltrated with fat.

Inguinal glands.—Vessels much congested; some in capsule contained micrococci.

CASE 24.—*Thyroid gland.*—Capillaries plugged with micrococci.

Lung.—Bronchial tubes in many places with epithelium detached or altogether gone, and with lumen filled with pus. In some parts lymph-follicles in walls of tubes. Extravasations of blood into air-vesicles; catarrhal pneumonia; capillaries in air-vesicles plugged with micrococci. In other parts air-vesicles filled with pus. Sub-pleural lymphatics in parts much distended and their walls thickened, so as to form distinct projections above the surface of the pleura. At other parts fibrous vegetations.

Heart.—Pericardium much thickened from swelling of fibrous tissues and considerable cellular infiltration. In some parts miliary abscesses. In other parts, vessels plugged in varicose form with micrococci. Intermuscular substance of heart infiltrated with cells so as in some places to greatly obscure muscle structure. Mitral valve normal.

Liver.—Thickened connective tissue of intralobular veins and cell infiltration of portal sheaths; bile ducts nearly occluded by swollen epithelium; liver-cells much pigmented, and capillaries in many parts filled with micrococci.

Supra-renal capsule.—A group of capillaries together with parent trunk, all plugged with micrococci. No other changes observed.

Kidney.—Epithelium swollen. Many tubes plugged with mulberry concretions and others with homogeneous coagula staining deep violet with hæmatoxylin. Numerous miliary abscesses, which in some parts had completely destroyed all kidney structure. In others dilated Malpighian bodies and the tube leading from them were covered with pus corpuscles, so as by their shape alone to show what they had originally been. Many vessels filled with micrococci. One such in the centre of a small abscess.

Spleen.—Increase of adenoid tissue. Many vessels plugged with micrococci.

Lymphatic glands; lumbar and axillary.—Swollen, vessels thickened, some in lumbar gland filled with micrococci. Also an abundance of bright yellow pigment granules.

Case 25. *Pyæmia.* (Appendix II., Table VII., Group V., Case 9.)

Lungs.—Pleura much thickened; in parts great dilatation of lymphatics and rupture at surface of pleura. Micrococci in subpleural vessels and on surface of pleura. Congestion of blood-vessels with micrococci, either filling up lumen of tube, or as round masses in coagula of fibrin and corpuscles. In parts, catarrhal pneumonia with micrococci in disintegrating epithelium.

Liver.—In some parts liver-cells cloudy. Nucleus invisible; outlines of cell indistinct. In others, cells containing much pigment and separated by greatly distended lymph spaces. Many capillaries contain micrococci.

Cellular infiltration of portal sheaths. Great thickening of sustentacular structure.

Kidney.—Great thickening of intima of larger vessels, smaller vessels with micrococci. Swelling of matrix near papilla, epithelium detached at many parts, at others swollen and cloudy.

Coagulum from femoral vein contained groups of micrococci. No other part preserved for examination.

Case 26. *Pyæmia.* (Appendix II., Table II., Group *a*, Case 19.)—Portions of almost all the organs of this subject, including some of the skin over the forehead where a swelling had been observed during life, were examined. In prostrate, bladder, and kidney were evidence of inflammatory changes to a considerable extent, both old and recent. The subcutaneous structure of forehead was œdematous, and infiltrated with round cells, but no micrococci could be found. Cf. Case 17.

Case 27.—*Pyæmia with multiple suppuration.*

Lung.—Purulent pneumonia; extravasations of blood. Some vessels plugged with laminated fibrin, a few micrococci seen in coagula in vessels, others in disintegrating cells and air-vesicles.

Liver.—Very considerable thickening of connective tissue round intralobular veins. Cell infiltration of portal sheaths, often to a considerable degree, and round small bile ducts, of which, however, the epithelium was normal, and the lumen, if anything, dilated. Liver-cells, some infiltrated with fat, others much pigmented, frequently shrivelled and disintegrated, round intralobular vein, and in some parts replaced by groups of small round cells.

Kidney.—Matrix near papilla much swollen and granular; basement membrane throughout more distinct than normal, and taking blue stain with hæmatoxylin. Epithelium unusually well preserved. Some blood-vessels plugged with coagula. Malpighian capsule and commencement of tubules often filled with homogeneous deeply-stained coagulum. A very few capillaries contained micrococci, and some larger vessels had small irregular patches of coagula adhering to the intima, and containing micrococci.

Spleen.—Adenoid tissue extensively replaced by fibrous tissue; in some parts altogether, in others much less. Other parts quite normal. Extravasations of blood.

Case 28. *Necrosis of femur; secondary abscesses in lungs.*—Male, æt. 54. *Post-mortem* twenty-eight hours after death.

Kidney.—Some of the veins near surface of kidney were so enormously distended that not more than about half could be got into field of microscope with No. 4 object, Hartnack. These vessels were some filled with laminated fibrin, others with grey-coloured débris, which with No. 11 immersion, Hartnack, was seen to consist of very minute uniform granules, perhaps débris of decomposing blood. Smaller vessels had similar débris intermixed with fragments of laminated fibrin. In centre of kidney some vessels with laminated fibrin adhering to walls of very irregular thickness. In one Malpighian body a single vessel of the glomerulus was enormously distended with bacteria. The vessel had ruptured, and the bacteria had escaped into cavity of capsule, and then expended into a cauliflower-like mass (Plate X. fig. 1). The matrix was

much swollen, and in many places stained violet. The basement of convoluted tubes was much thickened, stained blue, and in some parts where the epithelium had fallen out showed processes such as were described in case 20, but much thickened and deeply stained (Plate XI., figs. 3 and 4). Epithelium throughout very granular and detached. Specimen not in good preservation.

In many of the Malpighian bodies there was laminated coagulum between glomerulus and capsule. In others no trace of structure could be made out, all being replaced by a homogeneous, deeply-stained violet mass. Many of the convoluted and straight tubes contained similar material.

Axiliary vein.—Filled with coagulum. Two bands of fibrin extended across lumen from wall to wall, dividing vessel into three compartments. The central contained fine bands of fibrin radiating from the limiting bands and many colourless corpuscles, the lateral compartments only coloured corpuscles. Walls of vein much thickened. Vasa vasorum congested, and some containing micrococci. Tissue round vein much infiltrated with small round cells.

Axiliary gland. — Vessels in both capsule and gland substance much thickened. Those in gland substance usually homogeneous in appearance and deeply stained. Some micrococci in vessels.

Thyroid gland.—Changes in vessels similar.

Cases 29, 30, 31.—These were so much alike that one description may do for all:—Catarrhal pneumonia, infarcts, extravasations, thickening of pleura, interstitial inflammation of heart, liver, and kidney, fibrous changes in spleen.

Case 30.—Bacteria were found in a medium-sized vessel in heart, and in a glomerulus of kidney; but their position, free in the lumen of the otherwise empty vessel, suggested the possibility of *post-mortem* origin.

All these cases, but more especially 30, showed swelling of matrix, which reached even to free border of kidney in some places. It had a ground-glass appearance. Many of the glomeruli appeared as homogeneous violet-coloured masses.

41 showed in liver extensive fatty degeneration, but sometimes curiously limited to a single cell. Thus, in a section, a cell might be seen composed of a narrow ring of protoplasm, and a large cavity representing where the dissolved fat had been. Such a cell would be separated from any similar cell by several comparatively normal cells. In other parts a group of cells would be seen so changed. Micrococci were found in liver and kidney of this case and not in the others.

Jeremiah McCarthy.
W. S. Greenfield.

Description of Plate I.

Fig. 1. Forms of bacteria found in the blood during life. Drawn to scale, with the same magnifying power.

a. Extremely minute-jointed filaments.
b. Rod-shaped moving bacteria.
c. Ovoid spore-like bodies.
d. Clusters of similar ovoid bodies.
e. Micrococci.

Fig. 2. Section of heart of case of myositis (Appendix IV., Case 2) after staining with methylaniline and treatment with acetic acid. About 300 diam.

a. Muscular fibres.
b. Intermuscular connective tissue distended with exudation containing bacteria (only the bacteria are seen, the tissues being rendered transparent by the acetic acid).

Fig. 3. Arteriole in the heart from same case. Under the same magnifying power.

m. Muscular coat.
i. Intima. Swollen probably by action of reagents.
b. Colony of bacteria in vessel.

Fig. 4. Capillary venule in the tissues beneath an operation wound containing masses of micrococci. (The surrounding tissues are not represented).

a, *a'*, *a''*. Masses of micrococci deeply stained with methylaniline violet.
a. Masses more or less loose in channel.
a'. Masses closely adherent to the wall.
a''. Mass mingled with blood-corpuscles. Magnified about 200 diam.

Fig. 5. Micrococci in spleen.

a. Mass of micrococci deeply stained.

Fig. 6. Mass of micrococci in inflamed muscle (see also in Plate II., fig. 1).

a. Fibrinous exudation containing leucocytes.
b. Mass of micrococci deeply stained. 350 diam.

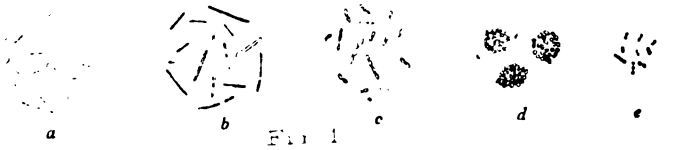


Fig. 1

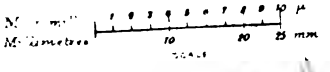


Fig. 2

Fig. 3.

Fig. 4

Fig. 5

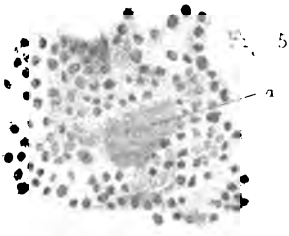
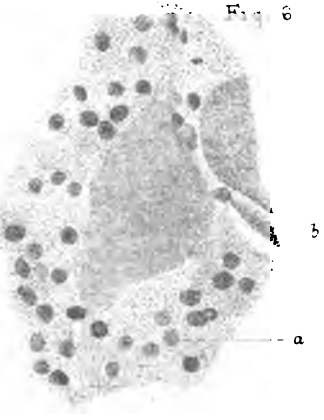


Fig. 6



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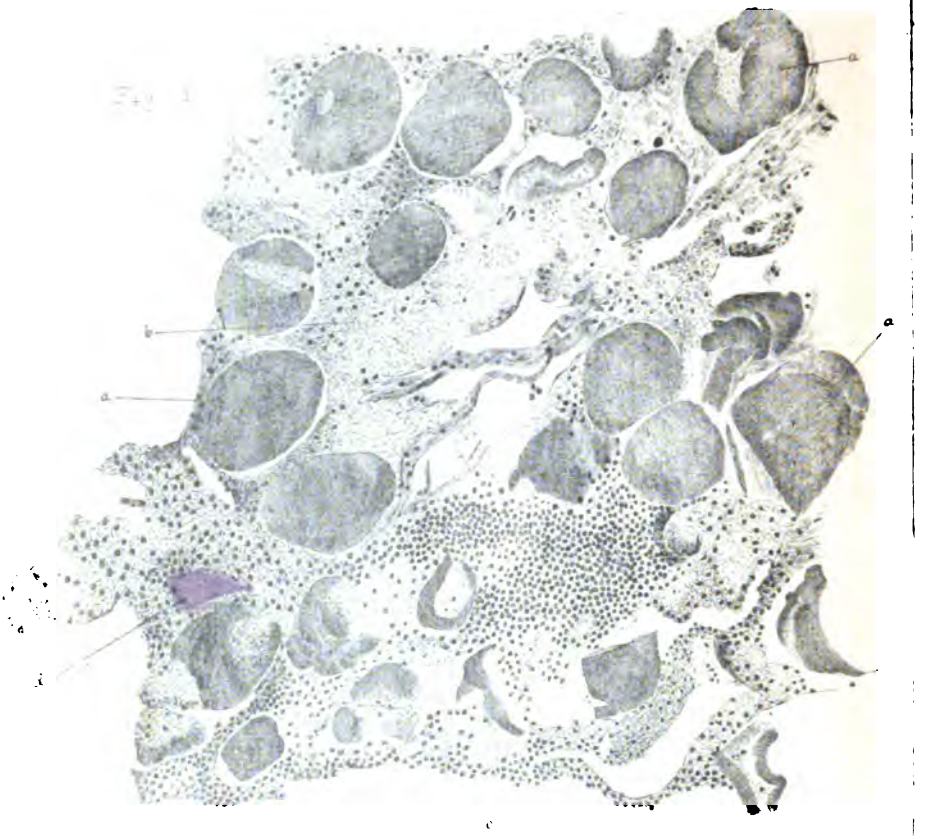

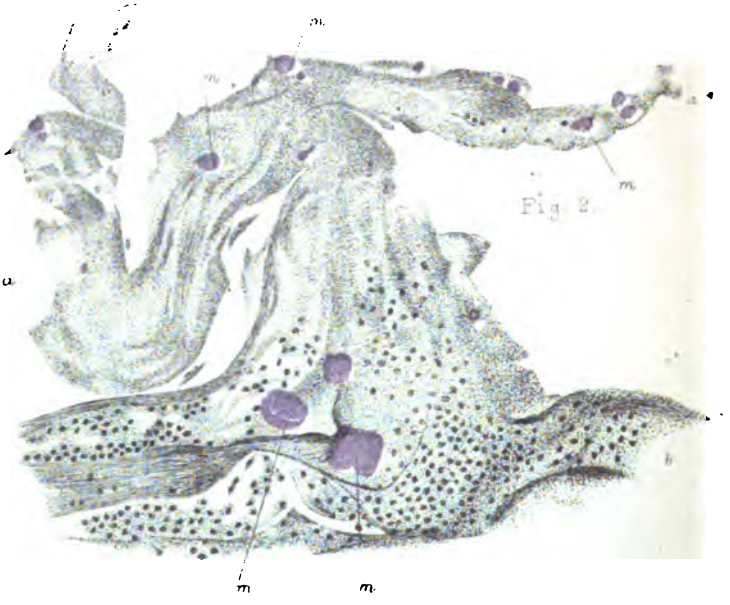

Fig 2.

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Description of Plate II.

Fig. 1. Inflamed muscle from forearm in myositis. 80 diam.

a. Muscular fibres.

b. Fibrinous exudation separating muscular fibres.

c. Exudation consisting in great measure of leucocytes.

d. Mass of micrococci (more highly magnified in Plate I., fig. 6).

Fig. 2. Fibrinous exudation in pericarditis, containing micrococci (treated with acetic acid). 180 diam.

a a. Layer of fibrinous exudation on surface, containing masses of micrococci (*m*).

b b. Superficial layers of pericardium, containing masses of micrococci, some in vessels (*m'*).

m m'. Masses of micrococci deeply stained.

Description of Plate III.

Fig. 1. Micrococci in pericarditis (from same case as Plate II., fig. 2, but under lower power). Treated with acetic acid after staining. 45 diam.

a a. Swollen superficial layers of pericardium.
b b. Masses of micrococci.
c. Venule containing commencing thrombus.

Fig. 2. Arteriole in tissues beneath a wound, showing swelling of inner coat. 45 diam.

a. Outer coat.
b. Middle coat.
c. Inner coat, much swollen.

Fig. 3. Arteriole in the skin adjacent to a minute abscess in a case of chronic pyæmia. 120 diam.

a. Outer coat.
b. Muscular coat.
c. Intima swollen considerably.

Fig. 4. Plug of leucocytes in vein in lung.

a. Arteriole filled with blood.
b. Wall of vein.
c. Mass of leucocytes and fibrin.

Fig. 1.

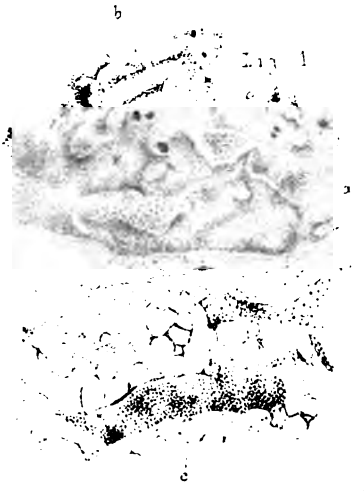

Fig. 2.

Fig. 3.

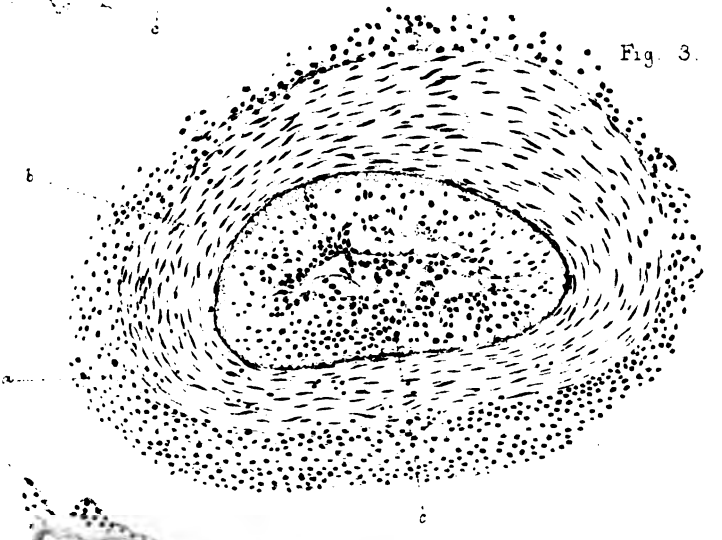

Fig. 4.

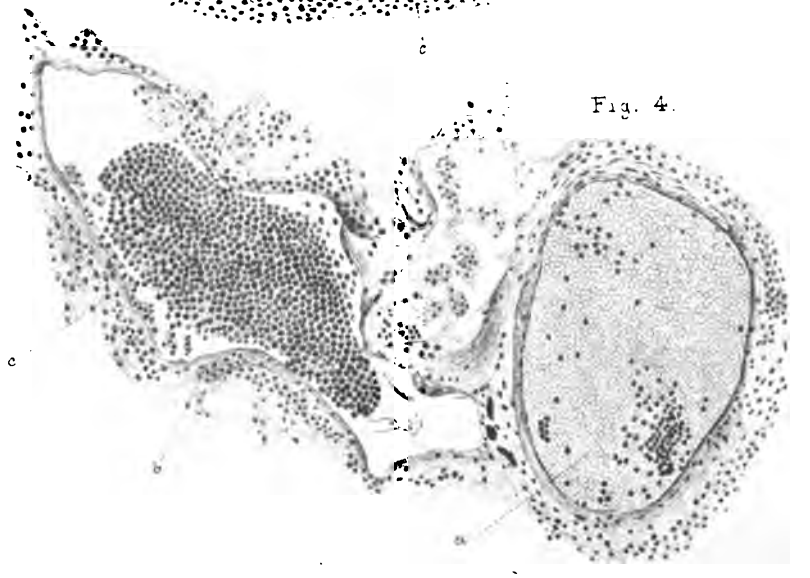

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Fig. 1

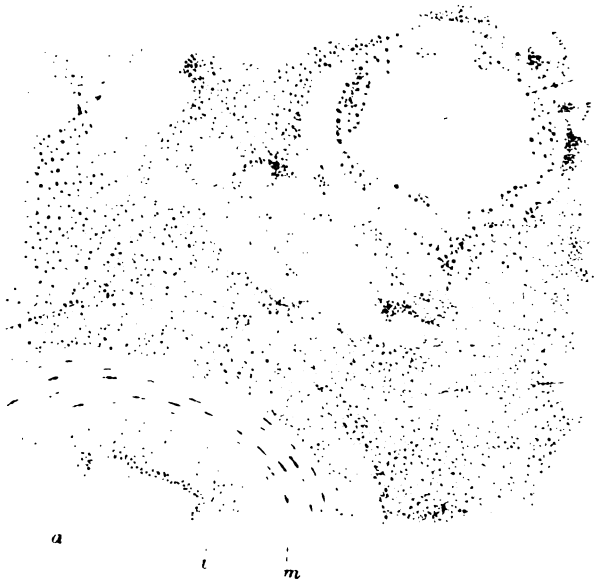
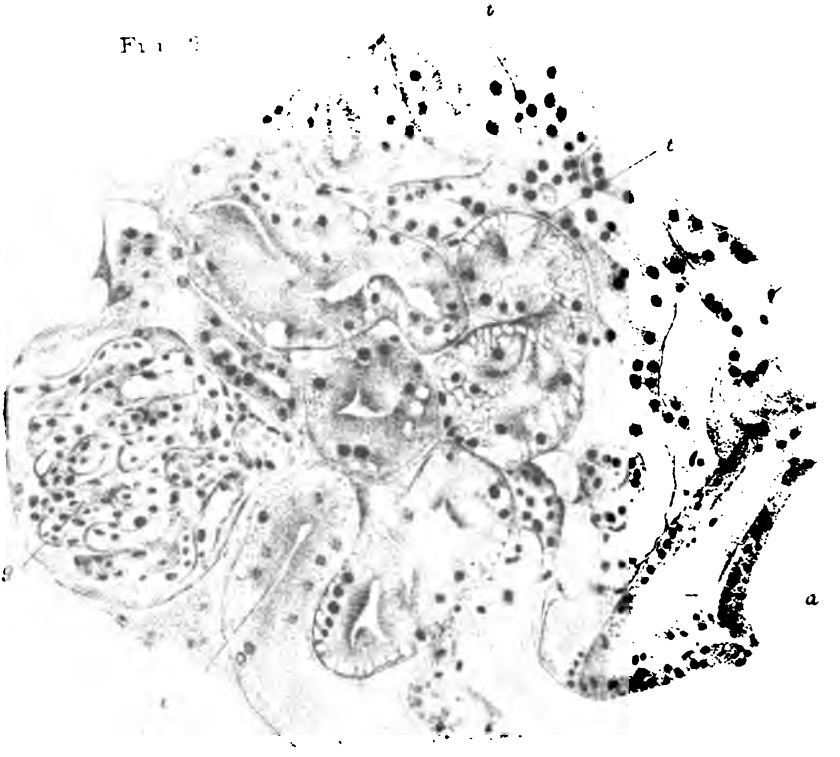


Fig. 2



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Description of Plate IV.

Fig. 1. Part of an abscess in subcutaneous tissue in a case of chronic pyæmia. 60 diam.

a. Arteriole. *m*. Its muscular coat. *i*. Intima.
v. Venule, almost entirely destroyed.
c. Contents of abscess.

Fig. 2. Kidney in septicæmia. 200 diam.

a. Artery.
g. A Malpighian body.
t t'. Convoluted tubules, the epithelium of which is swollen, with a sort of vacuolation in the superficial part and fragmentation in some parts, *t'*.

Description of Plate V.

Fig. 1. Liver in septicæmia (seen with low power). 25 diam.
v c. Central veins of lobules.
p v. Portal venule in portal space, with artery and bile-duct.
i. Interstitial exudation at periphery of lobules.

Fig. 2. Liver in septicæmia (same as fig. 1). Part of lobule near periphery, seen under a higher power. 350 diam. Hepatic cells swollen, fused together, and either segmented or breaking down.

Fig. 3. From the same, near centre of lobule. Hepatic cells swollen, nearly obliterating capillary network, each cell having an appearance of segmentation of the protoplasm, which in some is clear, in others cloudy, as in the normal condition. The network in the nuclei is also well seen.

Fig. 4. From the same ; showing somewhat similar appearances.

Fig. 5. Minute abscess in the brain. 45 diam.

Fig 1

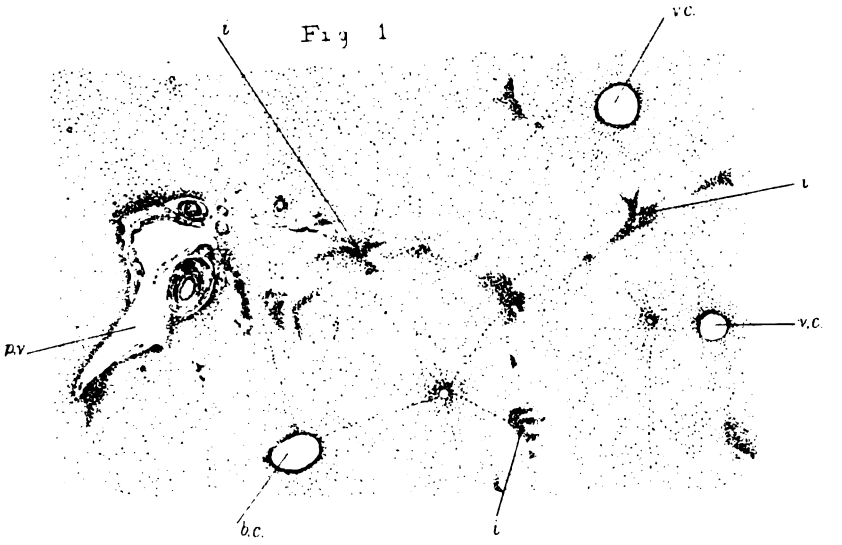

Fig 2

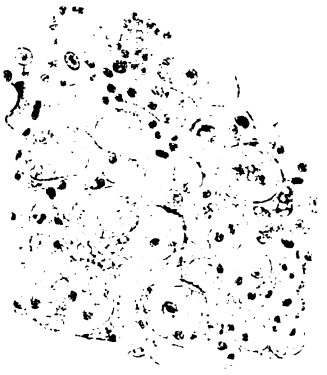

Fig 3

Fig 4

Fig 5

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Fig. 1

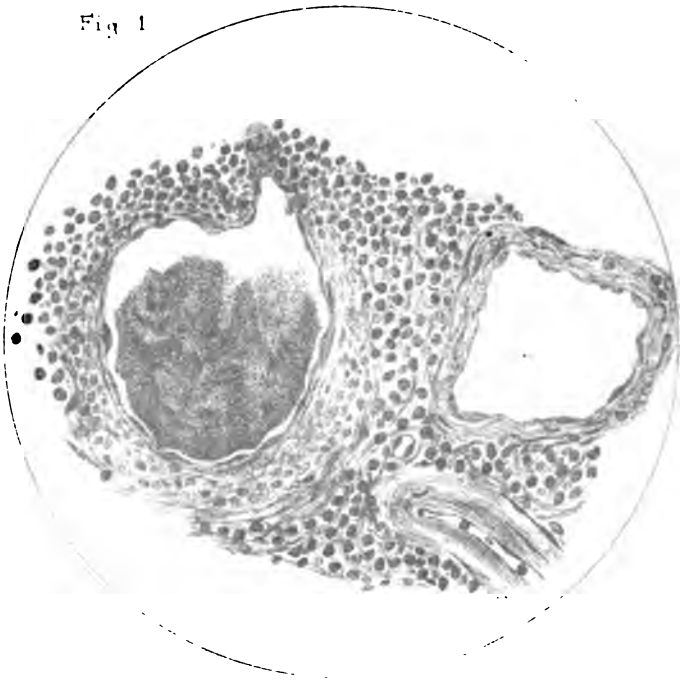
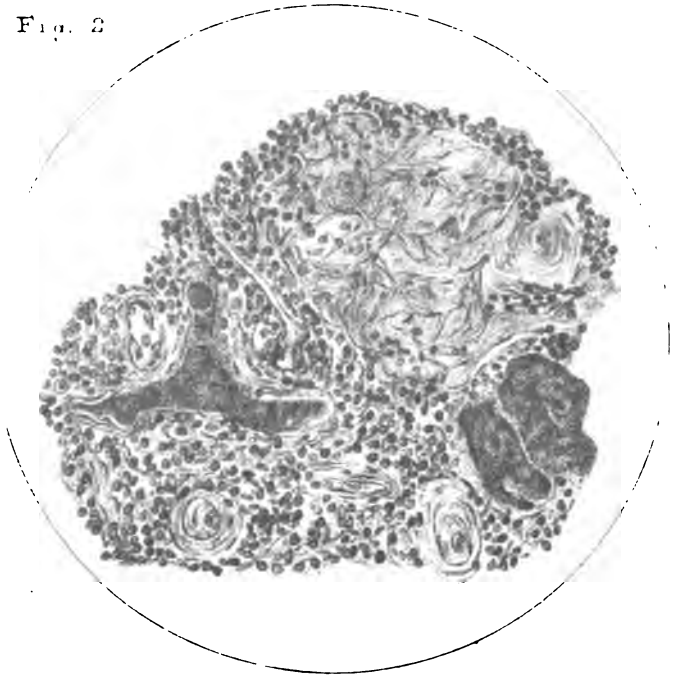


Fig. 2



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class of sugars of which cane sugar and sugar of milk are representatives. Cerebrose resembles sugar of milk by its feebly sweet taste, and the great hardness of its crystals.

D. *Properties of Cerebrose from pure Phrenosin.*—The crystallised cerebrose is never obtained without the amorphous modification being formed at the same time. To judge from comparison of bulks, at least half the cerebrose obtained during the chemolytic operations on the nitrogenised principles passes into the amorphous state, and cannot be made to crystallise entirely even within the period of a year. Crystalline particles of pure cerebrose immersed in the syrup increase in size, and form ramifications, but their growth ceases after a time, and the syrup thereafter remains unchanged. The difficulty of separating the two modifications is increased by the occasional appearance of a third form of product of the chemolytic metamorphoses of the amylonide radicle of the nitrogenised bodies which I shall presently describe.

The crystallised cerebrose obtained from pure phrenosin had all the properties of that described above. It was repeatedly attempted to ascertain more accurately the reducing power of the cerebrose over potassio-cupric tartrate such as is used in the process of analysis bearing the name of Fehling; in another series of experiments over sodio-mercuric cyanide, the reagent first recommended by Liebig and studied more particularly by Knapp (Ann. Chem. 154, 252). The experiments with the cupric solution confirmed those previously arrived at, viz., that a quantity of cupro-potassic tartrate which requires five parts of dextro-glucose for complete reduction requires about six parts of cerebrose; while those with the mercuric solution did not lead to any acceptable theory at all. Indeed, during these tests on the reducing power of cerebrose, I had to experience most of the irregularities of the tests alluded to, which have since been so fully described by Soxhlet (Journ. Pract. Chem., 21 (1880), 217–318). The application to cerebrose of the corrections shown to be requisite in the analysis by these methods of common sugars, such as grape sugar, or dextrose, invert sugar, lævulose, milk sugar, and maltose was impracticable, as the processes by which alone these corrections are attainable require larger quantities of material than were at my disposal.

Cerebrose is precipitated from its watery solution by basic lead acetate or by a mixture of neutral lead acetate and ammonia; the mother-liquor of this precipitate no longer reduces potassio-cupric tartate, from which it may be inferred that the precipitation of the cerebrose is complete. The lead compound of cerebrose after decomposition by hydrothion yields the cerebrose in the free state. In its affinity for lead oxide cerebrose resembles inosite, the sugar naturally contained in the brain and obtained as an educt from the water extracts (see Reports, New Series, No. VIII. (1876), p. 143), but it is easily distinguished from inosite by its power over polarised light and potassio-cupric tartrate, reactions which inosite does not possess.

E. *The Uncrystallised Cerebrose.*—The uncrystallised cerebrose obtained from the nitrogenised substances by the process of chemolysis above described may be a product from the crystallised; at least when the watery solution of the sugar is evaporated in the open air on the water-bath no crystallised sugar is ever obtained, but, as previous experience and renewed experiment have shown, only uncrystallisable cerebrose. Only when the solution is evaporated in a vacuum ensuring the absence of air, and at a temperature never rising above that of the animal body, 37°, is crystallisable cerebrose obtained,

accompanied, however, always by a considerable proportion of uncrystallisable cerebrose, amounting in weight to about that of the crystallisable cerebrose. There is therefore room for an inquiry into the causes of these phenomena. It may be that the uncrystallisable is only a derivate of the crystallisable cerebrose produced by the influence of air and heat; it may, however, also be that they are equivalent cleavage products of a sugar such as cane sugar, which latter splits up into a crystallisable dextro-glucose, and an uncrystallisable lævo-glucose.

The chemical constitution of cerebrose now arises as a subject of inquiry of interest and importance. Not only is there a new isomer added to the long list of saccharoid substances already known, but what is of much greater value, a new key is found to the knowledge of the constitution of some of the organo-plastic substances. This will enable us to obtain a full knowledge of the constitution of the nitrogenised substances of the brain much more quickly than would be the case without such theoretical aid. For the number and nature of the problems are now at once limited and defined, as we shall see presently by the aid of further new data.

F. *Cerebrosic Acid.* $C_6H_{10}(H_2)O_8$.—This new acid, obtained by means of the chemolytic process from phrenosin, has the composition of a carbohydrate, and is probably isomeric with cerebrose. It has not yet been examined any closer in the free state, but the examination of its barium salt leads to the inference that it is a dibasic acid of the formula $C_6H_{12}O_6$. It was obtained as follows: 25 grm. of pure phrenosin were suspended in 300 c.c. of water, and to the mixture 2 c.c. of oil of vitriol were added. The whole was enclosed in the platinum chemolyser (described on p. 137 of Reports, New Series, No. VIII., 1876), and heated to 120° during seven days without interruption. The acid liquid from the chemolyser was now filtered and treated with barium carbonate at boiling heat. The filtrate (which in the previous chemolyses of phrenosin, where the dilute sulphuric acid was renewed every 24 hours, mainly contained cerebrose) was found not to have any reducing effect at all upon potassio-cupric tartrate, but to contain a considerable amount of barium in solution. When evaporated quickly to dryness it left as residue a hard amorphous barium salt, but when dissolved in a little spirit and allowed to stand, it slowly set into a mass of indistinct crystals. These were freed from mother-liquor by pressure between folds of bibulous paper, dried at 102° C., and analysed with the following result:—

Barium Cerebrosate. Analysis.—(*a.*) 0·2862 grm. burnt after previous addition of some sulphuric acid gave 0·2116 grm. $BaSO_4$, corresponding to 43·50 per cent. Ba.

(*b.*) 0·3318 grm. burnt in vacuo with potassic dichromate, cupric oxide and copper gave 0·2983 grm. CO_2, and 0·0958 grm. H_2O, corresponding to 25·43 per cent. of carbon and 3·20 per cent. of hydrogen:

Elements.	Per centages.	÷ At. Wts.	÷ Ba = 1.
C	24·53	20·442	6·40
H	3·20	3·20	10·09
Ba	43,50	0·3175	1·00
O	28 77	1,798	5·66

These data lead to a formula $BaC_6H_{10}O_6$, corresponding to an acid of the composition of cerebrose in which two atoms of hydrogen are replaced by an atom of barium. It reminds one of glucic acid, first described by Peligot in 1838 (Ann. Chim. Phys. 67, 154) and further examined by Mulder (Journ. Pract. Chem. 21, 229), which is also dibasic. Glucic

acid is prone to form bodies like the caramels, and the presence of a small proportion of one or other of these bodies seems to be the cause of an excess of carbon over the theoretical amount, which is met with in the analysis of its salts. A similar feature is observed upon the cerebrosate of barium, which also exhibits a slight excess of carbon and a deficiency of hydrogen and oxygen. The salt is free from nitrogen, and does not contain sulphuric acid in organic combination. When distilled with phosphoric anhydride it does not give out the odour of acrolein, but a smell resembling burnt sugar. With oil of vitriol alone it gives no purple reaction. Mixed with sphingosin and oil of vitriol it gives a brilliant purple reaction equal in tint to that produced by the aid of cerebrose. But even its concentrated solution has no influence on the alkaline copper solution.

H. *Transformation of Barium Salt into Zinc Salt.*—The barium salt was decomposed with dilute sulphuric acid, and the solution extracted with ether. The ether, on distillation, left an acid which was converted into zinc salt; the latter was crystallised and analysed. The air-dried salt lost 9·8 per cent. of water at 100° C. The remaining dry salt was burnt, and the zinc estimated, from the remaining oxide, to amount to 28 per cent. of air-dried salt. From these data it seems that the zinc salt is entirely different from any of the known lactates. The barium salt, though different in appearance from lactate, was isomeric with it.

I. *Attempt to produce Cerebrosic Acid from free Cerebrose.*—A consideration of the conditions under which this acid had been produced from phrenosin, suggested that it might have been formed by the prolonged action of heat and acid upon cerebrose formed during the earlier stages of the reaction. In the experiments in which cerebrose was obtained, the influence of acid and heat upon the phrenosin was interrupted every 24 hours, and the acid was renewed. In the experiment, however, which yielded cerebrosic acid, the action of acid and heat had been continuous during seven days and nights. It was therefore necessary to investigate whether cerebrose already formed could be transformed into cerebrosic acid under the circumstances related, or whether it could be so transformed only in the nascent state, and what were the other conditions of this transformation.

About 2 grms. of amorphous cerebrose were heated with water containing one per cent. of oil of vitriol, to 120° during nine days. There were formed, firstly, a considerable amount of caramel; and secondly, a quantity of an acid corresponding, as regards its properties and those of its salts, to cerebrosic acid. But a large proportion of the cerebrose remained unchanged. The caramel was, like the acid, soluble in ether, and this so increased the difficulties of the separation from each other of these products, obtained necessarily from only small quantitites of material, that the separation was not completed.

K. *Caramel obtained in the Chemolysis of Phrenosin in which Cerebrosic Acid was formed.*—Of this caramel 2·1 grm. were obtained. It was very soluble in ether, and somewhat soluble in alkaline water, but insoluble in alcohol and in acidulated water. It was of a deep brown colour, like all the bodies of this class. From a consideration of the several forms of caramel which are obtained from the several principles of the nitrogenised group, as will be shown in another chapter, it becomes probable that the caramel here obtained was in part, at least, the caramel of psychosin. This supposition is strengthened by the consideration of the relative quantities of the several products of the chemolysis of phrenosin. The 25 grm. of this body employed in the experiment related above, gave 4·6 grm. of cerebrosic acid. The

theoretical amount which could have been obtained in the best case would have been 6·2 grm. Consequently 1·6 grm. of the amylonide radicle must have been transformed into bodies other than cerebrose and cerebrosic acid. The 2·1 grm. of caramel found in the present experiment could not have been caramel of cerebrose simply, as the atomic weight of that body is much smaller than that of cerebrose. But it could have been, or contained, some caramel of psychosin, the atomic weight of which is more than twice that of cerebrose. The other products insoluble in water were, sphingosin, which as sulphate weighed 2·5 grm., corresponding to about 2·13 grm. free sphingosin, and an unascertained quantity of another alkaloid, probably psychosin, which remained in the alcohol from which the sphingosin was precipitated by sulphuric acid. The amount of free fatty acid obtained was 9·4 grm. This was almost entirely neurostearic acid, and its quantity came very near to that required by theory, which is 9·9 grm. The total of weighed products of chemolysis amounted to 18·2 grm.; the psychosin or second base was not weighed, and allowing for this, certainly a few grm., and much for loss in the many difficult manipulations, the fate of the phrenosin originally employed is pretty well accounted for.

In almost all chemolyses of nitrogenised principles by acids or alkalies in watery or spirituous solution there has been formed cerebrose, cerebrosic acid, psychosin, caramel of psychosin, sphingosin, and neurostearic acid or its ether. When the cerebrose solution was freed from sulphuric acid, after chemolysis with an acid, it always retained some baryta (see the observation in Reports, New Series, No. VIII. (1876), p. 40) which had to be removed by precipitation with sulphuric acid. It is therefore possible that the cerebrose is always accompanied by some cerebrosic acid. Attempts should be made to extract this by ether before evaporating the cerebrose solution to a small bulk, as otherwise the acid may contribute to transform the cerebrose into the uncrystallisable modification.

V.—The Products of the Chemolysis of the Nitrogenised Principles insoluble in the Dilute Sulphuric Acid.

A. *Sphingosin, a new Alkaloid as Sulphate, and Fatty Acids.*—The solid products from the chemolytic tubes were united, edulcorated with water, dissolved in hot spirit, decolorised with animal charcoal, crystallised and dried. In a state of fine powder they were extracted with pure ether in the cold. The fatty acids dissolved, while a body remained insoluble, which was of an alkaloidal nature, and to which in commemoration of the many enigmas which it presented to the inquirer, I have given the name of *Sphingosin*.

Sphingosin Sulphate.—The part insoluble in ether was again treated with alcohol, but the substance, previously freely soluble in spirit, now became more and more insoluble, and at last fused and became quite insoluble even in absolute alcohol. It was now easily soluble in benzol in the cold. Addition of any acid to the hot alcohol restored the solubility of the body, and on cooling the body crystallised again. This bearing left little doubt that the body was a salt-like combination of an organic base with the acid employed, soluble in alcohol in the presence of an excess of acid, insoluble in the absence of such excess.

B. *Removal of the Sulphuric Acid by Caustic Alkali.*—The salt was freed from spirit by water, and then while diffused in water was treated with caustic soda ley and heated. The flaky body at once transformed into oily drops which rose to the surface of the liquid. On cooling, this oily liquid did not set like a fat but became again opaque,

and distributed in flakes through the fluid. The oil was, however, easily soluble in pure ether (in which the salt had been previously insoluble) and was extracted by this solvent. The solution was filtered, the ether distilled off, the residue dissolved in absolute alcohol and decolorised by animal charcoal. Minute quantities of impurities—probably of soda soap—which deposited from the ether solution and from this last alcoholic solution on standing, were removed by filtration. This alcoholic solution of the free base, which was alkaline to test paper, gave the following reactions. Oil of vitriol in absolute alcohol gave an immediate white precipitate of a sulphate. Hydrochloric acid gave a precipitate of a hydrochlorate; both precipitates were soluble in cold alcohol with the aid of excess of acid. An alcoholic solution of cadmic chloride gave a precipitate soluble in excess of absolute alcohol. Mercuric chloride gave a flaky precipitate which settled easily. Water caused a gelatinous precipitate in the alcoholic solution. Ether or alcohol solution left the base in a crystalline state on evaporation. It was very slightly if at all soluble in water, even on boiling When dry it gave no purple colour with oil of vitriol alone on gentle warming, but on addition of sugar gave immediately a deep purple colour. The full significance of this reaction will be discussed lower down.

C. *Sphingosin Sulphate.*—A solution of the free base in cold absolute alcohol was precipitated with a freshly made solution of oil of vitriol in absolute alcohol, with the precaution of keeping the alkaloid in excess. The white crystalline precipitate was washed with absolute alcohol, pressed and dried in vacuo over sulphuric acid.

Analyses.—(1) 0·3972 grm. fused with nitre, &c., gave 0·1404 grm. $BaSO_4$, equal to 14·56 per cent. SO_4; (2) 0·2636 grm. gave 0·0902 $BaSO_4$, equal to 14·09 per cent. SO_4, mean 14·32 per cent. SO_4 (3) 0·2127 grm. burnt with lead chromate, potassium chromate, cupric oxide, and copper in vacuo gave (*a*) 0·2049 grm. H_2O=10·70 °/₀ H; (*b*) 241·88 c.c. carbonic anhydride, normal, equal to 60·85 °/₀ C; (*c*) 7·04 c.c. Nitrogen normal = 0·0088 grm. or 4·14 °/₀ N.

These data lead to the formula for sphingosin sulphate of 2 ($C_{17}H_{35}NO_2$) + H_2SO_4, of which the theory is compared with the experimental data in the following table:—

Theory of Atoms.		Theory of Per cents.	Found. Per cents.
34 C	408	60·11	60·85
72 H	72	10·78	10·70
2 N	28	4·19	4·14
4 O	64	—	—
S	32	14·37	14·32
4 O	64		
	668		

D. *Sphingosin Hydrochlorate.*—On adding to a concentrated solution of sphingosin in absolute alcohol or in spirit some hydrochloric acid, a turbidity or precipitate is at once produced. If the mixture is warmed and allowed to cool gradually under a dryer over oil of vitriol, masses of spear-shaped crystals form in the fluid which can be removed as a felted mass. The crystals under the microscope appear as uniform needles with pointed pyramidal ends. The analytical data concerning this salt show that its formula is $C_{17}H_{35}NO_2$ + HCl. It does not easily form double salts with metallic chlorides.

The purification of sphingosin is based upon its precipitation from absolute alcohol by sulphuric acid and the decomposition of the sulphate in water by caustic alkali; the oily mass when freed from all alkaline liquid can be boiled with water, and thus freed entirely from alkali. In this way two impurities which may accompany sphingosin in small quantities are entirely removed; one a body soluble in ether, and which will be described hereafter; another an acid of which also a small quantity escapes the first extraction by ether, in which the bulk of the acids formed in the chemolysis is separated. Both remain in the absolute alcohol from which the sphingosin is precipitated by sulphuric acid. But they are less soluble in watery alcohol than the sphingosin, and therefore are precipitated by water or watery reagents together with the sphingosin.

E. *Consideration of the general Chemical Function of Sphingosin.*—The consideration of the molecular formula of sphingosin might at first sight lead to the hypothesis that it contained a fatty acid radicle of the $C_n H_{2n} O_2$ series, and that in this an atom of hydrogen was replaced by the amide group NH_2, as expressed by the formula $C_{17}H_{33}(NH_2)O_2$. Sphingosin also behaves like an amido-acid in this, that on the one hand it combines with bases such as potash, or on the other, unites with acids such as sulphuric and hydrochloric. However, its qualities as an acid are the least apparent, and so limited that they subsist in any degree only in the absence of water. In the presence of water the potash compound is not formed at all: the barium compound is formed in the presence of water and excess of barium salt, but decomposes during every treatment for its purification, even by resolution in strong spirit. The salts of sphingosin with acids, however, are very firm compounds: they crystallise, do not dissociate in solvents, and in the dry state admit of convenient manipulation. By its greater affinity for acids, sphingosin indeed differs from the amido-acids and resembles more the alkaloids. It is precipitated by most of the specific reagents which combine with alkaloids, and therefore on the whole evidence it must be admitted that sphingosin is an alkaloid.

F. *Neutral, Acid, and Basic Salts; Bearing of the Sulphate.*—When sphingosin sulphate is treated with dry neutral ether it remains undissolved. When the mixture is acidified with sulphuric acid the sulphate dissolves completely. When to the solution some alkali is cautiously added, the neutral sulphate is again precipitated in flocks. When neutral sphingosin sulphate, which has been dried completely, is digested in the cold with aqueous ammonia, sulphuric acid is dissolved and appears in the filtrate. But it is not practicable to remove all the sulphuric acid from the sphingosin by this treatment. Even after many days washing the alkaloid retains some sulphuric acid. This can only be removed by warming the compound with caustic ley and extracting the free alkaloid with ether.

It follows from the foregoing that sphingosin forms neutral and acid, and perhaps basic salts.

G. *Bearing of the Hydrochlorate.*—The hydrochlorate of sphingosin crystallises from water or alcohol in long, spear-shaped cystals. It is much more soluble in hot water than in cold; it is deposited from a solution in water which also contains psychosin almost entirely if the solution is cooled to and filtered at the temperature of melting ice. A little psychosin, however, easily remains with the sphingosin, so that in analyses of the latter the carbon is sometimes found a fraction per cent. too high. The admixture is recognised by the purple test with sulphuric acid, and is removed by recrystallisation of the hydrochlorate,

or in case of the sulphate by transformation into the free base, and reprecipitation from absolute alcohol by sulphuric acid. The psychosin salts of both acids are the more soluble ones.

H. *Sphingosin with Potash.*—When the solution of free sphingosin, prepared from the pure sulphate in ether, is dried by being allowed to stand over solid caustic potash, a dense but translucent deposit in the shape of flakes and crusts covering the sides of the vessel and the masses of potash is gradually formed. This is a compound of sphingosin with potash which is little soluble in the anhydrous ether. It has not yet been separated from the excess of potash. The ether retains a certain portion of this compound in solution, and leaves it as a hard, dense, colourless deposit on distillation. A preliminary analysis of this residue gave 61·04 per cent. carbon, 9·96 per cent. hydrogen, 4·52 per cent. nitrogen, and 6·53 per cent. potassium. From this it is evident that this residue is a mixture of a compound formed of sphingosin and potassium or potash, with free sphingosin. The potassium compound $C_{17}H_{34}KNO_2$ requires 12·1 per cent. of potassium. The mixture therefore contains a little more than half its weight of the potassium compound.

I. *Separation of Psychosin from Sphingosin.*—This may be effected in alcoholic or watery solution. The mixed alkaloids are dissolved in absolute alcohol and a very dilute solution of oil of vitriol in absolute alcohol is added so as to precipitate all sphingosin. From the filtered alcoholic mother-liquor all alcohol is removed, first by distillation, afterwards by evaporation with water. The residue is warmed with some caustic potash, and the ley which has taken up any sulphuric acid is decanted. The washed residue is boiled with hydrochloric acid, in which it dissolves readily. The solution is filtered hot and concentrated to a suitable bulk. It contains all the psychosin, and if the precipitation of sphingosin by sulphuric acid had been incomplete, or an excess of the acid had been added so as to redissolve some sphingosin as acid sulphate, this base also occurs in the hydrochlorate. The solution, on being cooled to 0° C., deposits sphingosin hydrochlorate in colourless crystals, which may be filtered off in the cold, but cannot be washed from excess of psychosin by water, as they swell and make filtration impossible.

K. *Fatty Acids and Matters soluble in Ether being Products of the Chemolysis which yielded Sphingosin.*—The total quantity of these bodies ultimately obtained weighed 48·7 grm., while the total of the sphingosin sulphate obtained from the same chemolysis weighed 40·4 grms. No great importance is attached to these relative weights, first, because the matters chemolysed were known to be mixtures, secondly, because much matter was unavoidably lost in the countless operations to which the tubes and their contents were subjected, and in the numerous quantitative and qualitative tests to which the products had to be subjected for the eliciting of information concerning the new radicles. Nevertheless the numbers bear out the theory of the nitrogenised substances in a general way which will be made more apparent in the future.

The ether solution was concentrated, and yielded several deposits of fatty matters at several stages. The first deposit after recrystallisation from spirit resembled neurostearic acid in appearance. It began to fuse at 73°, but did not wholly fuse till the temperature reached 81°. The body is therefore probably neurostearic acid, mixed with a small quantity of acid of lower fusing point. It was converted into baryum salt; this was extracted with hot spirit, and the fatty acid was again extracted from the baryum salt by tartaric acid and ether. The free acid now fused at 80° and set at 79°; it had therefore lost some of the fatty acid melting at a lower temperature.

The later deposits from ether and all the acids most soluble in ether were converted into baryum salts and exhausted with boiling alcohol. There remained insoluble in spirit a baryum salt, which by treatment with tartaric acid and ether gave an amount of free fatty acid (19 grm.), which fused below 50° C., and therefore differed greatly from the acid first deposited from the ether solution.

The alcoholic extracts of the baryum salts on cooling deposited a *solid compound*, and retained in solution a compound which, after evaporation of all alcohol, was *semi-solid* on being heated with water, and dissolved entirely in ether.

We have therefore obtained four principal bodies by these operations on the chemolysed matters soluble in ether. Two form baryum salts insoluble in boiling spirit, and are, the first mainly neurostearic acid, $C_{18}H_{36}O_2$, first described on p. 288 of the Report of the Medical Officer of the Local Government Board for 1877, the other an acid of lower fusing point, not yet studied any further. The bodies soluble in hot spirit, and of which one was soluble in ether, correspond to the bodies already mentioned on pp. 287 and 289 of the Report just quoted. The significance of all these bodies will be made clear when we come to consider the chemical constitution of the cerebrin-like substances. Each of these acids, which by their number no less than their properties are remarkable, will then find a place in a natural educt, and by another process in a systematic classification.

VI.—Psychosin, its Properties and Metamorphoses.

A. *Comparison of Sphingosin and Psychosin.*—On p. 136 of these Reports, New Series, No. VIII. (1876), I have described a new alkaloid obtained from the nitrogenised bodies termed cerebrins, by chemolysis with caustic baryta. The mere analysis of this body led to an empirical formula of $C_{25}H_{49}NO_8$, which was uncontrolled by combinations. The free body had, however, crystallised from alcohol. To fill up this void I produced some combinations with hydrochloric acid and with platinic chloride, but the products had the peculiarity of some of the phosphorised bodies, viz., of dissociating in the presence and under the influence of small quantities of water. The hypothetical salt would have contained 24·01 % of platinum, whereas the actual salt contained only 21·05 %. But the carbon of the organic molecle decreased slightly in relation to the nitrogen, and this led me to again analyse a further purified specimen of psychosin.

B. *New Analysis of Psychosin.*—The psychosin was dissolved in hydrochloric acid and water, and the solution made perfectly bright and colourless. The alkaloid was then precipitated by ammonia, washed perfectly, and dried in vacuo.

Analyses.—(*a.*) 0·6027 burnt with cupric oxide and copper gave 0·5475 grm. H_2O and 1·3531 grm. CO_2, equal to 10·093 % H and 61·32 % C. (*b.*) 0·3310 gave 85 c.c. Nitrogen gas at Bar. 755·6 K H O column = 81 mm. T = 11° 5 C, equal to 3·04 % N.

Summary of Analyses of Psychosin.

	First Analyses (1876).	*Present Analyses* (1878.)		
	Per-cents.	Per-cents.	At. Wts.	÷N = 1.
C	61·86	61·32	5·11	23·53
H	10·09	10·09	10·09	46·46
N	2·88	3·04	·21716	1·00
O	25·17	25·55	1·597	7·35

C. *Psychosin Sulphate*.—Psychosin is soluble in very dilute sulphuric acid on boiling, but is deposited on cooling. The mother-liquor of the deposit after 24 hours retains only a small amount of the salt in solution, so that ammonia, which, if not added in excess, precipitates psychosin from its solution in water and hydrochloric acid, hardly causes any precipitate. But phospho-molybdic acid causes a more appreciable precipitate.

D. *Psychosin Hydrochlorate*.—When to a solution of neutral psychosin hydrochlorate in water an excess of strong hydrochloric acid is added, a bulky gelatinous precipitate, much resembling hydrate of alumina, is produced. The salt is so completely removed from the solution that the latter, after filtration, gives no precipitate with phospho-molybdic acid.

When a solution of psychosin hydrochlorate is kept on a dialyser of parchment paper floating on distilled water for 18 hours, some psychosin, as well as some hydrochloric acid, passes into the water. The amount which passes is very small and no psychosin is deposited on the dialyser. Psychosin is therefore a strong base, but at the same time it exercises its functions as a colloid, such as it becomes in the presence of water and absence of acids.

Psychosin precipitated from its hydrochlorate solution by ammonia and well washed, when allowed to remain in contact with pure water during several days, becomes hydrated and swells up to a voluminous gelatinous mass. This paste retains water with great force, and is most difficult to dry in vacuo over sulphuric acid. Heat causes it to become brown at once; even in the vacuum it assumes colour.

E. *Psychosin and Ammonia*.—Psychosin dissolves readily in concentrated ammonia water on boiling, and is deposited again on cooling and standing. The hot ammonia solution gives a precipitate with barium chloride, which after isolation is soluble in boiling alcohol and deposited on cooling. This compound loses barium by all attempts at re-crystallisation.

F. *Chemolysis of Psychosin by Dilute Sulphuric Acid*.—One grm. of the psychosin which had been analysed was enclosed in the platinum chemolyser with a sufficiency of sulphuric acid of 2 per cent. strength, and heated to 130° during 40 hours. The sulphuric acid solution was separated from the insoluble part, boiled with barium carbonate, &c., and tested. It reduced a quantity of Fehling's solution, which if the sugar had been glucose would have indicated 0·173 grm. of it. The solution also contained a small amount of an acid similar to the cerebrosic acid described in the present report. The cerebrose obtained amounted therefore to less than half the theoretical quantity, but was supplemented by a small portion of its derivate.

The solid products of the chemolysis were coloured brown; when warm they were in a state of semifusion. The mass was gently warmed with soda solution and shaken with ether. It dissolved in the ether without residue, imparting to it a deep brown colour. The ether was distilled off and the residue treated with hot absolute alcohol. This solvent left a small quantity of caramel of pyschosin as a brown mass, and dissolved a quantity of slightly coloured matter. The solution was decolourised with animal charcoal, and precipitated with a little sulphuric acid dissolved in absolute alcohol. The precipitate was isolated, washed with ether, and dried. It gave no purple reaction with oil of vitriol alone, thus proving the absence of psychosin, but with a solution of cane sugar or cerebrose, the sulphuric acid solution gave a brilliant purple reaction. The precipitate was therefore sphingosin sulphate.

The alcoholic mother-liquor of the sphingosin sulphate contained some matter in solution, which gave, after removal of the alcohol, a purple reaction with oil of vitriol by itself. It was some psychosin which had resisted the chemolytic action of the dilute sulphuric acid.

The chemolysis of phychosin by sulphuric acid therefore takes place according to the equation—

$$\underbrace{C_{23}H_{45}NO_7}_{\text{Psychosin.}} + H_2O = \underbrace{C_6H_{12}O_6}_{\text{Cerebrose.}} + \underbrace{C_{17}H_{35}NO_2}_{\text{Sphingosin.}}$$

G. *Purple reaction with Oil of Vitriol.*—I have shown in various parts of these researches, *e.g.*, p. 302 et seq. of Report for 1877, that all the nitrogenised bodies of the brain, or cerebrins, give with oil of vitriol, on gentle warming, a reaction, which consists in the formation of a deep purple colour. This reaction, which when produced with cane-sugar and oil of vitriol, is known as Pettenkofer's reaction, and was for a long time believed to be specific to biliary acids, has been shown to be common to a large number of bodies which probably have a radicle in common. But most of these bodies, *e.g.*, oleic acid, require the addition of sugar, and do not give the purple with oil of vitriol alone. Now all the cerebrin bodies are distinguished from the biliary and fatty bodies by the faculty of giving the purple reaction under two different sets of conditions. They give it with oil of vitriol alone, on gentle warming, after a little time of standing; and they give it more quickly if with the oil of vitriol a little sugar is at once added. Now we know that all these bodies contain the radicle of the sugar cerebrose, which, as I have shown on p. 140 of Report, New Series, No. VIII., (1876), gives with pure glykocholic acid and sulphuric acid a brilliant Pettenkofer reaction, and is consequently capable of replacing cane-sugar to its full value in this process. It is therefore clear that the ability of the cerebrin bodies to give the purple with oil of vitriol alone has for one of its causes the presence in their constitution of the radicle of cerebrose. That they react more slowly with sulphuric acid alone than with sulphuric acid and sugar added, is explained by the facts evolved in the chemolysis above described, namely that the splitting off of cerebrose from the other radicles requires time.

We are now able to make the hypothesis—which has a high degree of probability—that every cerebrin-like body which gives a purple reaction with oil of vitriol alone contains the radicle of cerebrose besides that other radicle which with any sugar, cerebrose, or cane-sugar, gives the purple, and which we will term the oleo-cholide radicle. On the other hand, any cerebrin body which does not give the purple with oil of vitriol alone may contain either the cerebrose or the oleo-cholide radicle. If, on addition of sugar, it gives the purple, then it contains the oleo-cholide radicle: if it does not give the purple, then this radicle also is excluded.

I will now proceed to the application of these data to the testing of the chemolytic products above described. Sphingosin, with oil of vitriol at a very gentle heat, dissolves and becomes a little yellow. But no purple colour is produced. On addition of cane-sugar or of cerebrose in concentrated solution the purple is immediately struck. Psychosin, with oil of vitriol at a very gentle heat, becomes yellow and brownish while dissolving, and then the purple colour appears without any addition of sugar. Consequently there is a *primâ facie* presumption that psychosin still contains the cerebrose, while from sphingosin it is detached. We have already seen how well this presumption, derived from the chemical reactions of these bodies, is supported by their relative chemical

formulæ. The reaction can consequently be used for demonstrating the purity or impurity of any specimen of sphingosin as regards its freedom from or contamination with substances capable of yielding cerebrose with oil of vitriol. The substances most likely to remain mixed with sphingosin in small quantity are those of which it is a cleavage product, and more particularly psychosin, which like sphingosin is an alkaloid.

H. *Isolation of the Purple Products.*—The purple bodies produced in the reactions of the cerebrin products described in the foregoing pages are, as I have already shown in the Report for 1877, p. 302, under certain conditions soluble in chloroform; it is necessary to place the mixture in bottles carefully stoppered, and keep them anhydrous by excess of oil of vitriol. The clear dry chloroform solution can be distilled from dry vessels boiling, and leaves the purple product behind. The residue dissolves in new chloroform with a finer purple colour than before and completely. A little water added to the purple chloroform solution makes it turbid, and destroys the colour completely in a few minutes. The purple which has been re-dissolved after the removal of the first chloroform by distillation is destroyed by water instantaneously.

I. *Intermediate Products of Chemolysis of the Cerebrin Bodies with Sulphuric Acid, Hydrated Phrenosin, Æsthesin, Psychosin.*—We know from previous experience that the decomposition of the cerebrins by baryta ensues in a much shorter time than by acids. We also know that the products, though essentially the same in both processes, are differently distributed, and are split off at different times and in a different order. It is essential to know all those products which are intermediate, in the first instance, because without them the formulæ of the decomposition cannot be made evident with all necessary detail, and secondly, because small quantities of these intermediate products mostly outlast the chemolytic process, and then occur as impurities in the final products or are left as residues incapable of purification and analysis.

K. *Preliminary Hydration.—Splitting off of Cerebrose.*—In the First Report on the Brain (Reports, New Series, No. III., 1874, p. 189), I have described a preliminary experiment with sulphuric acid and spirit on phrenosin. In this a product was obtained which, to judge from the changes in the per-centage composition, was simply hydrated.

	Original Phrenosin.	*Same after short Chemolysis.*
	Per cents.	Per cents.
C	66·12	64·791
H	10·85	11·077
N	2·26	1·960
O	20·74	22·172

This corresponds most nearly with the hypothesis that a molecle of water had entered the molecle of phrenosin, while no sugar certainly, and no other large molecle probably had been detached. The change is in harmony with what chemical theory would lead us to expect, namely, a *preliminary hydration.* The hydrated product is soluble in ether, in which the phrenosin is insoluble.

The next step in the sulphuric acid chemolysis is the *splitting off of the cerebrose.* This, in the water solution, ensues very slowly, while the first hydration, to judge from the solubility of the product in ether, is more quickly attained.

The remainder of the radicles, minus the cerebrose, do yet hold together for a longer time before they split up into sphingosin,

fatty acids, and other bodies. Unfortunately, I have not been able to follow this second step upon the specimen of phrenosin employed in the hydration experiment just alluded to. But it is represented by the product of another cerebrin body.

Forty-four grms. of mixed cerebrins were hydrated by boiling in water, and pressing through a cloth. To the homogeneous paste 40 c.c. of oil of vitriol, already diluted with the amount of water necessary to prevent overheating, were added. The mixture was now boiled, and curdled immediately. Boiling was continued for nearly an hour, when the liquid containing much sugar, and becoming more coloured, heat was withdrawn.

The *curdled cerebrin matter* was transparent and soft while hot, slightly coloured red, and became solid and white immediately on cooling. In order to remove any fatty acids which it might contain, it was, after removal of all sulphuric acid, suspended in ammonia water, and precipitated by barium chloride. The curdy precipitate was found to be almost entirely soluble in hot spirit, and to contain hardly any fatty salt insoluble in spirit.

The spirit solution filtered hot through a heated funnel immediately deposited *white crystals*. These, under the microscope, were seen to consist of two bodies, one in needles, another in crystalline balls. The deposit from spirit was isolated and suspended in ether (2 litres).

A *body dissolved* that crystallised from the evaporating ether in the often-seen forms, namely, apparently curved needles, which will presently be more closely defined. Another body remained insoluble in ether, and on combustion left some baryta. This latter will not be considered any further in this place.

L. *Crystallised soluble in Ether Product.—Æsthesin.*—Six litres of ether were required for its complete extraction from the other products. The ether solution was distilled to a small bulk, and then allowed to crystallise. Over-night it formed a voluminous white mass consisting entirely of crystals, which were now uniform and showed many angular plates. They were collected on a filter and drained of mother-liquor by stirring. While wet they fused on the water-bath in the mother-liquor adhering to them, but became completely dry without much discolouration and were solid and waxy, therefore not fusible below 90°. Re-crystallised slowly from a dilute solution in ether, the crystals are seen to be hexagonal plates, more or less regular, but whether or not the six angles of the hexagons are equal cannot be determined. The plates are further saucer-shaped, and this produces the appearance of the curved or sickle-shaped needles when the bodies are seen sideways and the lower edge is out of focus. The plates are scarcely visible when they lie flat on the glass. They are distinctly recognised with all the details when they are made to roll edgeways over the field (Chinese hats).

The dried crystals dissolve in oil of vitrol and assume a yellowish but no purple colour. Cane sugar added to this produces the purple reaction soluble in chloroform and yielding the spectrum described and figured in Report for 1877. Consequently, this body contains the oleo-cholide radicle, but not the cerebrose radicle. It was now analysed. Between 80° and 150°, 0·1825 grms. lost in three stages 5 mgrm. in weight; the remainder burnt left 1 mgrm. incombustible residue. The dry powder fritted a little, and at 82° to 83° became a waxy transparent mass. It fused to a liquid at 110°, but did not become opaque again until cooled to 71°. By re-crystallisation its nitrogen, which at first was 2·70 °/₀, could be depressed to 2·35. After purification and crystallisation from absolute alcohol and from pure ether, in both of which it was soluble in the cold, it crystallised in the same hexagonal

plates. Heated to 150° it became coloured and exhaled an odour of burnt fat. For analysis it was heated to 100° for three hours.

Analyses.—(*a.*) 0·4310 grm. gave 9·8 c.c. of gas (nitrogen). H col. = 63 m.m. KHO col. = 183 m.m. Bar = 742·5. T = 14° C.; = 2·35 °/₀ nitrogen.

(*b.*) 0·4354 grm. gave 0·4890 H_2O = 12·73°/₀ H, and 1·1947 CO_2 = 74·83 °/₀ C.

Summary.

	Per cents.	÷ At. Wgts.	÷ N = 1.
C	74·83	6·24	37·14
H	12·73	12·73	75·79
N	2·35	·168	1·00
O	10·09	·631	3·75

These results prove that æsthesin is an intermediate product of cerebrin chemolysis; it has lost the cerebrose radicle, as is evident from the low quantity of oxygen it contains, and it still retains the oleocholide radicle, as is evident from its yielding the purple reaction when sugar is added. It therefore behaves like a sphingosin to which a fatty acid radicle is still combined. The nitrogen is a little too low, that is to say, æsthesin is already mixed with a trace of fatty acid, but the oxygen stands to carbon in the relation of 4 atoms to 35 atoms. Hence the hypothesis that æsthesin is a compound of sphingosin and neurostearic acid gains a high degree of probability, thus:—

Sphingosin + Neurostearic acid = Æsthesin. Water.

$$\overbrace{C_{17}H_{33}NO_2} + \overbrace{C_{18}H_{36}O_2} = \overbrace{C_{35}H_{67}NO_3} + \overbrace{H_2O}$$

If now to æsthesin we add a molecle of cerebrose, we obtain—

Æsthesin + Cerebrose = Cerebrin – Body. Water.

$$\overbrace{C_{35}H_{67}NO_3} + \overbrace{C_6H_{12}O_6} = \overbrace{C_{41}H_{77}NO_8} + \overbrace{H_2O}$$

The fatty acid radicle contained in æsthesin is supposed to be neurostearic acid. The cerebrin body is very similar to the one described on p. 192 of the Report, New Series, No. III., 1874, the empirical formula of which is given as $C_{42}H_{83}NO_{10}$.

VIII.—Chemolysis of Pure Phrenosin by Sulphuric Acid in Alcoholic Solution; Formation of Ethylic Neurostearate.

A. *The Process.*—65 grm. phrenosin were suspended in 1500 c.c. of spirit of 85 °/₀ strength and 200 c.c. of oil of vitriol gradually added while the mixture was well agitated. The hot mixture, on which an oily matter floated, was then boiled for 2½ hours in such a manner that the volatilised alcohol was condensed and ran back again into the fluid. On cooling the oily layer solidified, and a slight deposit formed in the underlying fluid. The insoluble and deposited parts were separated by filtration from the acid solution and dissolved in ether; the ethereal solution was agitated with dilute solution of caustic soda, by which a small quantity of fatty acid was removed as soda salt. The clarified ether solution was distilled, and the residue was repeatedly crystallised from absolute alcohol. From this solvent it was deposited in small but well-marked shining crystals, which were very soluble in hot alcohol, but very little in cold. The recrystallised matter fused at 56° C., but the manner of its fusion seemed to indicate an admixture of a less fusible with a more fusible body. It was subjected to a preliminary elementary analysis by the vacuum method, with the following result:—0·0872 grm.

gave 0·1080 grm. H_2O, 0·2443 grm. CO_2, and 0·00043 grm. nitrogen. The small amount of nitrogen was evidently due to the presence of a small quantity of a nitrogenised substance which was at once eliminated by the process of distillation in vacuo about to be described. The recrystallised body was mixed with glass powder and placed in a glass tube closed at one end. This was then bent downwards at a point beyond the mixture, and a little further on bent again upwards, drawn out and connected airtight to a mercurial air pump. The air was then completely pumped out, and heat cautiously applied until the matter was distilled into the V-shaped part of the tube; this was effected without the evolution of any gas and without a trace of charring. When at the end of the distillation the slight residue in the glass powder began to discolour and to evolve gas, the operation was stopped.

B. *Neurostearic Ether*, $C_{20}H_{40}O_2$.—The distilled matter was of the colour and consistence of bleached beeswax. It melted at 52 °C. On analysis 0·2956 grm. were found to give 0·8312 grm. CO_2, and 0·3446 grm. H_2O, corresponding respectively to 76·69 % of carbon and and 12·95 % of hydrogen. It contained no trace of nitrogen. These numbers agree with those demanded by the formula $C_{20}H_{40}O_2$.

	Theory		Found.
	of atoms.	of per cents.	
C_{20}	240	76·92	76·69
H_{40}	40	12·82	12·95
O_2	32	10·26	10·36
	312	100·00	100·00

This body is ethylic neurostearate $(C_2H_5)\ C_{18}H_{35}O_2$, as was proved by the following experiment:—4·5 grm. of the ether, which had been completely freed from any adhering alcohol by fusion at 110° during several days, were enclosed in the platinum chemolyser with concentrated soda ley, and heated to 100° for eight hours, during which the chemolyser was frequently shaken. The caustic solution when quite cold was poured upon a filter of glass wool, and by this means the soap, which was quite insoluble in the concentrated ley, was separated from the excess of alkali; and, after rinsing with cold, was dissolved in a large quantity of boiling water. It gave a clear solution. A portion of this solution precipitated hot with barium chloride gave a barium salt containing 23·94 % of barium. This would indicate a monobasic acid of atomic weight 218. The remainder of the barium salt was converted into free acid, which was dried and dissolved in ether. On concentration, the ether gave a crystallisation of perfectly white neurostearic acid in a characteristic mamillary form. The ethereal mother-liquor crystallised similarly in cauliflower-like masses to the last drop. This acid fused at 84° C. It was dried by fusion at 100° C., and analysed. 0·1617 grm. gave 0·1840 grm. OH_2 and 0·4503 grm. CO_2, equivalent to 75·94 % carbon, and 12·64 % hydrogen.

	Theory.		Found.
Atoms.	At. Wts.	Per cents.	Per cents.
C_{18}	216	76·06	75·94
H_{36}	36	12·68	12·64
O_2	32	11·26	11·42
	284	100·00	100·00

The concentrated soda ley employed in this chemolysis of the neurostearic ether was examined for alcohol. It was distilled, the distillate enclosed hermetically in a glass tube with excess of chromic and sulphuric acids, and heated for six hours to 100°. The chromic acid was partially reduced, and on distillation of the fluid, an acid distillate was obtained. On boiling with barium carbonate, and evaporation to dryness, it gave 0·32 grm. dry barium acetate, giving a red colouration with ferric chloride, evolving the pungent fumes of acetic acid when moistened with oil of vitriol, and giving the fragrant odour of acetic ether with alcohol and oil of vitriol.

C. *Psychosin Sulphate.*— The alcoholic mixture of sulphuric acid and other decomposition products from which the neurostearic ether had been removed by filtration, was treated with caustic lime in powder, and the liquid, now free from sulphuric acid, filtered from the gypsum, and some calcium neurostearate which was mixed with the precipitate. The alcoholic solution was distilled, and after the alcohol had passed off, left a voluminous white pasty mass of free psychosin. This was dissolved in hydrochloric acid and ether, and thus separated from the impurities, of which sulphovinate of calcium was the principal one. The ether solution of psychosin hydrochlorate was freed from ether by distillation, the residual salt dissolved in water, and allowed to stand in the cold. It deposited colourless crystalline plates, masses of fine microscopic crystals of extreme tenuity, but showing geometric definition of sides and angles; these easily redissolved if the liquid was allowed to rise in temperature. Filtration was therefore effected in the cold. These crystals consisted of pure sphingosin hydrochlorate, as was proved by isolation, decomposition with caustic potash in the hot, extraction of the base with ether, solution of the base, after distillation of the ether, in absolute alcohol, and precipitation of the sulphate by oil of vitriol dissolved in absolute alcohol. The psychosin in the solution was identified by isolation, and all the characteristic tests. This experiment was the first one in which psychosin was obtained by chemolysis of phrenosin with an acid.

D. *Chemolysis of Phrenosin with a view to the Quantation of its Products.*—Several experiments were made to ascertain the proportions between phrenosin and its products of decomposition and the proportion of these products to one another.

(1.) Two grammes of phrenosin were enclosed hermetically in a glass tube with dilute sulphuric acid of one per cent. strength. The tube was put into the platinum chemolyser, surrounded with distilled water, and heated to 120° C. for 24 hours. The glass tube was then opened and the dilute acid removed. The sugar in this was determined by boiling a known proportion of it with excess of alkaline copper solution, dissolving the cuprous oxide in ferric chloride and sulphuric acid, and titrating the solution with potassic permanganate. By this means the amount of sugar was found to be 0·28 grm. A quantity of cerebrosic acid ($C_6H_{10}(H_2)O_6$) was also found. By estimating the amount of barium dissolved on boiling the fluid with baric carbonate, it was ascertained to amount to 0·072 grm. Fresh sulphuric acid of one per cent. strength was put into the glass tube and the heating continued for another 20 hours. The fluid then again reduced copper solution, and gave cuprous oxide equal to about 0·02 grm. cerebrose. These three quantities of carbohydrate added together amount to over 18 per cent. of the phrenosin employed. The theory given in another part of this report shows 25 per cent. as the utmost possible quantity. Considering the nature of the reaction, the quantity actually obtained is an approach to the theoretical amount, especially as it is shown in experiment II.,

described a little further on, that caramel, psychosin, and a little undecomposed phrenosin are also among the products of the reaction, and these, in all probability, contain the greater part of the missing 6 per cent. of cerebrose.

The solid products of the reaction were rubbed to a fine powder with cold water, washed free from adhering sulphuric acid, and dried in vacuo over oil of vitriol. The mass weighed nearly 1·7 grm. A portion was analysed for sulphuric acid by heating with caustic soda, nitre, and carbonate of soda, and precipitating the hydrochloric acid solution of the fused mass with baryum chloride. In this way 0·1536 grm. gave 0·0220 grm. $BaSO_4$, equivalent to 6·03 per cent. H_2SO_4. The products gave a purple colour with oil of vitriol alone. On treatment with ether they separated into a soluble portion consisting mainly of neurostearic acid with a little caramel, and into an insoluble portion consisting mostly of sphingosin sulphate with a little undecomposed phrenosin, and perhaps some psychosin sulphate. The separation was not sufficiently perfect for quantitative purposes.

(2.) Another experiment was made on a larger quantity and by a somewhat different method. 10 grm. phrenosin were chemolysed with acid of one per cent. in the platinum chemolyser for nine days, the dilute acid being removed and replaced by fresh every day. The cerebrose in the acid fluids was estimated and found to be a little less than one grm. The amount of cerebrosic acid was found to be not more than 0·15 grm. The solid product was dissolved or finely distributed in large volumes of boiling dilute ammonia water. To this solution while yet hot barium chloride was added in excess. The whole of the organic matter was precipitated so that the fluid on evaporation and incineration did not show more than traces of charring. The curdy precipitate was well washed and dried. It weighed 11·3 grm. It was then rubbed to a fine powder and exhausted with boiling absolute alcohol. The matter which was left undissolved weighed dry 6·36 grm., and consisted of a mixture of sulphate of barium, the sulphuric acid of which was derived from its previous combination with alkaloids, with neurostearate of barium. It was titurated with hydrochloric acid and water, and extracted ether. The latter extracted 2·6 grm. of neurostearic acid with traces of caramel. After exhaustion with ether the matter was boiled with absolute alcohol. The alcohol extract left 0·2 grm. of a matter which from its appearance and properties seemed to be phrenosin, and had therefore escaped chemolysis. The residue insoluble in alcohol and ether still contained some organic matter besides barium sulphate, and as there was no simple mode of extracting this, it was burnt. The residual inorganic matter now weighed 2·085 grm. It was extracted with hydrochloric acid, to which it gave up some carbonate with effervescence. The barium sulphate left undissolved in the hydrochloric acid weighed 1·76 grm., while that dissolved, after transformation into sulphate, weighed 0·32 grm.

If we assume that the barium of the 0·32 grm. had been in combination with neurostearic acid, it would indicate 0·78 grm. of this body The whole amount of neurostearic acid would thus be raised to 3·38 grm. If we further assume the sulphuric acid contained in the undissolved $BaSO_4$ (1·76 grm.) to have been in combination with sphingosin so as to form $(C_{17}H_{35}NO_2)_2H_2SO_4$, then the quantity of this salt would have amounted to 2·51 grm., and in it would have been contained 2·15 grm. of sphingosin. If on the other hand we assume the sulphuric acid to have been in combination with psychosin, the quantity of psychosin contained in the compound would have amounted to 3·38 grm.

The absolute alcohol extract of the barium compounds described deposited at once on cooling, and again on concentration and cooling, 0·75 grm. of a body which was soluble in ether, fusible at a gentle heat, and solid on cooling; it was not examined any further. The absolute alcohol solution which remained after the removal of these two precipitates was mixed with a solution of sulphuric acid in absolute alcohol as long as a precipitate was produced, an excess of acid being carefully avoided. The white deposit of sphingosin sulphate was washed with absolute alcohol and dried in vacuo. It weighed 2·5 grm. The alcoholic mother-liquor of this sphingosin sulphate left on distillation a residue which was treated with caustic ley and ether. The ether dissolved and after distillation left behind 1·27 grm. of a matter which by its reactions was recognised as psychosin.

VIII.—Isolation and Purification of Cerebrinic Acid.

A. *The Lead Salts.*—The insoluble precipitate formed during the purification of phrenosin by the lead treatment was exhausted with hot 85 °/₀ spirit to remove all phrenosin and kerasin. It was then dried, powdered, and extracted with cold benzol.

(1.) *Portion soluble in Benzol.*—The extract containing lead cerebrinate was concentrated to a small bulk, and was warmed with 85 °/₀ spirit, and a little hydrochloric acid. The acid was found to partially decompose the cerebrinic acid formed. The benzol extracts were therefore evaporated to dryness, and the dry residue of lead cerebrinate was powdered and suspended in 85 °/₀ spirit. The spirit was saturated with hydrothion while being gradually raised to the boiling point, and the lead sulphide filtered off. On cooling a white body was deposited, which consisted mainly of *cerebrinic acid.* This was collected on a filter, purified by recrystallisation from absolute alcohol, and dried in vacuo. It gave a weak purple reaction with oil of vitriol alone. Under the microscope it appeared to consist of small needles. It was soluble in hot benzol, and was thrown down on cooling as a gelatinous mass. It did not blacken at 100° C. Some preliminary elementary analyses of this body are given below.

(2.) *Portion insoluble in Benzol.*—This was dried and suspended in 85 °/₀ spirit, and the lead extracted as in the former case by hydrothion. On cooling a white body was deposited, which was recrystallised, collected, and dried. It gave the purple reaction only very feebly. It was dissolved in hot absolute alcohol, and again combined with lead. The compound was not precipitated by acetate only, but appeared on addition of ammonia on cooling. It could, however, be dissolved again on warming.

The mother-liquor from the recrystallisation of the free body, obtained before it was again transformed into lead salt, gave a precipitate with lead acetate, but none with cadmic or platinic chloride.

On analysis the lead salt gave phosphorus equal to 0·19 °/₀, and two estimations of lead gave respectively Pb. = 20·06 °/₀ and 19·4 °/₀.

B. *Preliminary Analyses of Cerebrinic Acid.*—(*a.*) 0·385 grm. burnt with CuO and Cu in CO_2 gave 4·63 c.c. of N normal = 1·5 °/₀ N.

(*b.*) 0·389 grm. burnt with CuO and Cu gave 0·948 grm. CO_2 = 0·25855 grm. C = 66·47 °/₀ C, and 0·3975 grm. OH_2 = 0·04417 grm. H = 11·355 °/₀ H.

(*c.*) 0·382 grm. burnt with CuO and Cu gave 0·946 grm. CO_2 = 0·258 grm. C or 67·54 °/₀ C, and 0·391 grm. OH_2 = 0·04344 grm. H or 11·37 °/₀ H.

(*d.*) 1·3715 grm. burnt with soda lime gave 0·366 grm. Am_2PtCl_6 = 0·022944 grm. N, and 0·0135 grm. Pt. = 0·00191 grm. N, making 1·68 °/₀ N.

—	*a.*	*b.*	*c.*	*d.*
Nitrogen °/₀ -	1·5	—	—	1·68
Carbon °/₀ - -	—	66·47	67·54	—
Hydrogen °/₀ - -	—	11·355	11·37	—

C. *Cerebrose by Chemolysis of Cerebrinic Acid.*—An alcoholic solution of cerebrinic acid was chemolysed by sulphuric acid; and the liquid portion separated by filtration from the solid products. The solution was treated with phospho-molybdic acid to remove all alkaloids, and then with baryta to remove excess of sulphuric acid and phospho-molybdic acid. The filtrate was freed from excess of baryta by carbonic acid and was concentrated to a small bulk. The sulphovinate of barium was then decomposed by sulphuric acid in the least possible excess, a quantity of water was added, and the mixture was boiled for a long time. Barium carbonate was then added to remove the sulphuric acid formed by the decomposition of some sulphovinic acid, and the liquid was filtered. Lead acetate and ammonia were then added, and the precipitate was filtered off. On testing the filtrate for cerebrose none was found. The precipitate, after being washed by decantation, was decomposed by hydrothion, and the lead sulphide filtered off. The filtrate was tested with Fehling's copper solution, but did not give distinct results owing to the dark colour which the mixture assumed. Knapp's mercuric solution, however, gave results indicating the presence of 1·3 grm. cerebrose in the whole of the filtrate.

IX.—Action of Heat upon the Nitrogenised Substances; Formation of Caramels.

A. *Caramel of Phrenosin.*—(1.) *Preliminary Experiments.*—Some preliminary experiments for the study of the action of heat upon phrenosin may be here again referred to (see Reports, New Series, No. III., 1874, p. 191). "0·5290 grm. lost on drying between 18° and 70° C. 0·0084 " hygroscopic water, and the remaining 0·5206 grm. were considered as " dry substance. After two hours' exposure to a heat of 97° C. in a " large air-bath it became slightly yellow, and had lost 0·0016 grm. " After three hours' exposure to 101° it had remained of the same colour " and weight. After two hours' exposure to 145° it had become very " dark, almost black in colour, and superficially fused, and had lost " 0·0080 grm. After heating to 158° during three hours it had become " thoroughly fused, and of a reddish, almost transparent aspect; it had lost " 0·0120 grm. in weight. After four hours at 177° C. it had become " blacker and less transparent, and gave out a faint odour of burnt meat; " it had lost at this stage 0·0317 grm. Thus the phrenosin had lost in " four stages 0·0533 grm., or 10·2 °/₀ in weight. After cooling it was " hard and brittle. On boiling a piece in absolute alcohol only a trifling " amount of matter dissolved, colouring the solution slightly yellow. On " the other hand it readily dissolved in ether; the solution had a dark " reddish brown colour." "A little of the phrenosin was heated in a test " tube over the naked flame. It fused, turned dark, and evolved water " with ebullition, similar to sugar, passing into caramel. The water, which

" condensed in the upper part of the tube had an acid reaction, and " reduced copper solution. The fused matter became hard on cooling, " was but slightly soluble in boiling alcohol, but readily soluble in ether ; " from the latter solution it was reprecipitated by alcohol."

Similar experiments were now instituted with a view of increasing the accuracy of results, and the knowledge concerning the constitution and function of phrenosin and the other nitrogenised bodies.

(*a.*) *First Experiment.*—Some phrenosin was dried in a current of air at 100° C., in a Liebig's drying tube placed in a paraffin bath, connected at each end with a chloride of calcium drying tube, one to dry the air before it entered the tube and the other to arrest the moisture from the phrenosin evolved during the operation. A drying tube filled with glass saturated with sulphuric acid served to prevent moisture from the water pump working back into the chloride of calcium tube.

The dry phrenosin weighed 1·216 grm. The temperature was raised to 150° C., a current of dry air passing meanwhile. On weighing the tube it was found to be still of the same weight, but the chloride of calcium tube had gained ·019 grm., a fact which can only be explained by oxidation of the phrenosin. At this stage the phrenosin became brownish-white in colour, but did not fuse. The temperature being now raised to 160°, the phrenosin melted into a brown mass, and on being weighed was found to have lost ·014 grm., the chloride of calcium tube having gained the same amount. The temperature was then rapidly raised to 200° C., but there was no longer a current of air allowed to pass through, in order to retard as much as possible the evolution of volatile matter. On cooling, air was passed through as before, and the tube on being weighed showed a loss of 0·054 grm., the chloride of calcium tube having only gained 0·050 grm. The temperature was now finally increased to 240° C., with the same precautions against escape of volatile matter as before. Volatile matters were now, however, evolved which slightly blackened the sulphuric acid; and on severing the connexions of the apparatus a smell of burnt sugar was emitted. The tube showed a loss of 0·080 grm., while the chloride of calcium tube had gained 0·056 grm.

Thus in the whole operation the tube had lost 12·17 %, while the chloride of calcium had gained 11·425 %. Taking the formula of phrenosin as $C_{41}H_{79}NO_8$, the molecular weight of it would be 713. The preceding figures would then show, taking the loss as 12·17 %, a loss of 4·82 molecles of water ; and, taking the gain as 11·425 %, a loss of 4·52 molecles.

(*b.*) *Second experiment.*—Some phrenosin was dried at 100° C., the weight of the dry body being 0·789 grm. The temperature of the tube was now raised to 200° C., and kept at that point for two hours, air being passed through only on cooling below 150°. No volatile matters were carried into the sulphuric acid tube, though a slight sublimation of a volatile matter on to the cooler parts of the tube took place. On weighing, the tube was found to have lost 0·0456 grm., and the chloride of calcium had gained 0·054 grm. The heating was now continued at 200° for four hours, to try the effect of time on the operation, air being passed through only on cooling. The loss was now 0·0335, and the gain 0·0365. The totals in per cents. were a loss of 10·14 % or 4·02 molecles, with a gain of 11·47 % or 4·54 molecles of water.

In both these experiments the greater portion of the caramel was soluble in ether; though in the first case not so perfectly as in the second, on account of the higher temperature to which the former was raised.

(*c.*) Tabular View of the Data concerning Caramel of Phrenosin.

Dry Amount.	Temp.	Absolute Loss.	% Loss.	Total % Loss.	Molecles.	Absolute Gain $CaCl_2$.	% Gain.	Total % Gain.	Molecles.
1·216	150°	.00	·00	—	—	·019	1·56	—	—
—	160	·014	1·15	—	—	·014	1·15	—	—
—	200	·054	4·44	—	—	·050	4·11	—	—
—	240	·080	6·58	12·17	4·82	·056	4·605	11·425	4·52
·789	200	·0465	5·89	—	—	·054	6·85	—	—
—	200	·037	4·25	10·14	5·02	·0865	4·62	11·47	4·54

B. *Caramel of Cerebrinic Acid.*—(*a.*) *First experiment.*—1·613 grm. of dry cerebrinic acid were heated to 210° C. in a current of air for one hour, a similar apparatus to that described under "caramel of phrenosin" being used. The tube lost 0·122 grm. or 7·55 %, and the chloride of calcium gained 0·103 grm. or 6·38 %. Calculated on a formula (hypothetical) of $C_{59}H_{113}NO_9$, and a moleclar weight of 979, these figures would give a loss of 4·1 molecles on the basis of the loss, and 3·47 molecles on the basis of the gain, taken respectively.

(*b.*) *Second experiment.*—1·221 grm. of dry cerebrinic acid were heated to 160° C. The substance melted completely, and lost 0·030 grm., the chloride of calcium gaining 0·033 grm. The temperature was now raised to 200° C., and the current of air was passed only when the tube cooled. The loss and gain respectively were 0·065 grm. and 0·056 grm. The total loss was thus 7·77 % or 4·227 molecles, and the gain was 7·30 % or 3·97 molecles of water.

(*c.*) *Third experiment.*—1·073 grm. at 150° C. lost 0·012 grm., and the chloride of calcium tube gained 0·019 grm. At this temperature the cerebrinic acid melted. At 200° C. it lost 0·0965 grm., and the chloride of calcium gained 0·080 grm. The total percentages were 10·12 % loss or 5·5 molecles, and 9·23 % gain or 5·00 molecles of water.

(*d.*) *Fourth experiment.*—0·779 grm. of dry cerebrinic acid at 145° lost 0·006 grm., and the chloride of calcium gained 0·0125 grm. At 180° the substance was completely melted, and lost 0·033 grm., while the chloride of calcium gained 0·037 grm. At 210°, the current of air being stopped as before until the tube had somewhat cooled down, the loss was 0·0535 grm., and the gain 0·030 grm. The totals lead to a per centage loss of 11·67 % or 6·347 molecles of water, and to a gain of 9·98 % or 5·43 molecles. Oxidation may, as in the former case, account for this.

These caramels were also almost entirely soluble in ether.

(*e.*) Tabular View of the Data concerning the Caramels of Cerebrinic Acid.

Dry Amount.	Temp.	Absolute Loss.	% Loss.	Total % Loss.	Molecles.	Absolute $CaCl_2$ Gain.	% Gain.	Total % Gain.	Molecles.
1·613	210°	·122	7·55	7·55	4·10	·103	6·38	6·38	3·47
1·221	160	·030	2·46	—	—	·033	2·70	—	—
—	200	·065	5·32	7·77	4·227	·056	4·60	7·30	3·97
1·073	150	·012	1·12	—	—	·019	1·77	—	—
—	200	·0285	2·66	—	—	·0200	1·86	—	—
—	200	·068	6·34	10·12	5·504	·060	5·60	9·23	5·02
·779	145	·006	·77	—	—	·0125	1·60	—	—
—	180	·033	4·24	—	—	·037	4·75	—	—
—	210	·0535	6·66	11·67	6·347	·030	3·63	9·98	5·43

C. *Caramel of Psychosin.*—(*a.*) *Experiment.*—0·3615 grm. heated to 110° C., in the same apparatus, as in the other cases, lost ·021 grm., and the $CaCl_2$ gained ·0225 grm. At this temperature the substance became brown and caked. It was then heated to 160° C. when it fused completely, losing ·0145 grm., the gain being at the same time ·0185 grm. At 210° C. it had lost ·021 grm., while the $CaCl_2$ had gained the same amount. On cooling, it split up into flakes, which were only partially soluble in ether. At 210° C. the current of air was stopped, as there was a slight deposit of volatile matter on the cooler parts of the tube. The current was resumed as the caramel cooled.

(*b.*) Tabular View of the Data concerning the Caramel of Psychosin.

Dry Amount.	Temp.	Absolute Loss.	°/₀ Loss.	Total °/₀ Loss.	Molecles.	Absolute $CaCl_2$ Gain.	°/₀ Gain.	Total °/₀ Gain.	Molecles.
·3615	110°	·021	5·81	—	—	·0225	6·22	—	—
—	160	·0145	4·01	—	—	·0185	5·12	—	—
—	210	·021	5·81	15·63	4·038	·021	5·81	17·15	4·43

D. *Remarks on the Caramels.*—The preliminary experiment on the action of heat upon phrenosin quoted from Reports, New Series, No. III., 1874, showed a loss of 10·2 °/₀, which is equal to 4·03 atoms of water on the formula $C_{41}H_{79}NO_8$. The maximum loss which phrenosin experiences in the experiments here described is similar in amount, but the water collected does not tally with it. It is therefore clear that some oxidation takes place in the substance under caramelisation; this hypothesis does not, however, completely explain the discrepancies. In future experiments the operation should be carried on in a current of neutral gas, such as hydrogen or nitrogen, or in carbonic acid.

X.—Theory of the Chemical Constitution of the Nitrogenised Principles of the Brain.

Although the average of the analyses of phrenosin leads to data from which the true formula of its composition can be derived, it is a great advantage to be able to prove the true formula by the aid of the formulae of the products of decomposition. If phrenosin had never been analysed by itself, its formula could be predicted by means of the synthesis of its cleavage products, according to the following calculation:—

1 molecle of sphingosin - - -	$C_{17}H_{35}NO_2$
+1 do. of neurostearic acid - -	$C_{18}H_{36}O_2$
+1 do. of cerebrose - - -	$C_6H_{12}O_6$
−2 molecles of water - - - -	H_4O_2
leave one molecle of phrenosin - -	$C_{41}H_{79}NO_8$

Arranged analytically the equation would be the following:—

$$C_{41}H_{79}NO_8 + 2(H_2O) = C_{17}H_{35}NO_2 + C_{18}H_{36}O_2 + C_6H_{12}O_6.$$

Of this phrenosin the atomic and percentic theory is the following:—

Theory			Found mean.
Of Atoms.		Of per cents.	Per cents.
41 C	492	69·000	67·957
79 H	79	11·080	11·426
1 N	14	1·963	1·997
8 O	128	17·957	18·696
	713	100·000	

This phrenosin, in the course of chemolysis, by taking up two molecules of water, increases its atomic weight from 713 to 749, which latter figure represents the sum of the atomic weights of the three products of decomposition above enumerated. 100 parts of phrenosin, therefore, become 105 parts of products. Of these 39·6 parts are neurostearic acid, 39·9 parts sphingosin, and 25·1 parts cerebrose.

If now, with the aid of this theory, we attempt to revise the theories formerly given of some compounds of phrenosin, retaining the analytical data, we obtain a greater harmony than before. Thus the nitrited phrenosin shows the following theory:—

Nitrited Phrenosin Nitrate $C_{41}H_{78}(NO_2)NO_8 + HNO_2$, or contracted, $C_{41}H_{79}N_3O_{13}$.

	Theory		Found in 100.
	Of Atoms.	Per cents.	
41 C	492	59·92	59·58
79 H	79	9·62	9·93
3 N	42	5·11	4·65
13 O	208	25·33	25·84
	821		

Neurostearic Acid is accurately distinguished from all similar known acids, by its high fusing point (84° to 85°), and its atomic weight is well fixed by its ethyl compound. But its salts are not very stable, nor very precise bodies, or are mixtures, so that it has hitherto proved impossible to use them for stoichiometric purposes. The theory of this acid is the following:—

	Theory		Found	
	Of Atoms.	Of per cents.	In crystallised Acid.	In Acid from Ether.
18 C	216	76·056	75·88	75·94
36 H	36	12·676	12·85	12·64
2 O	32	11·268	11·27	11·42
	284	100·000	100·00	100·00

The ethylic compound of this acid is a very precise body; it is *Neurostearic Ether* or ethylic neurostearate, and its theory is the following:—

	Theory		Found
	Of Atoms.	Of per cents.	Per cents.
20 C	240	76·92	76·69
40 H	40	12·82	12·95
2 O	32	10·26	10·36
	312	100·00	100·00

The dissolved formula is $(C_2H_5)C_{18}H_{35}O_2$.

Sphingosin is a strong base and gives the most precise compounds of any of the products of the decomposition of phrenosin. Its theory is the following:—

	Theory		Per cents. found in Sulphate Acid deducted.
	Of Atoms.	Of per cents.	
17 C	204	71·59	71·26
35 H	35	12·28	12·53
1 N	14	4·91	4·82
2 O	32	11·22	11·39
	285	100·00	100·00

Sphingosin Sulphate. $2(C_{17}H_{35}NO_2) + H_2SO_4$.

	Theory		Found.
	Of Atoms.	Of per cents.	Per cents.
34 C	408	60·11	60·85
72 H	72	10·78	10·70
2 N	28	4·19	4·14
4 O	64	10·55	9·99
1 S, 4 O	32, 64	14·37	14·32
	668	100·00	100·00

Cerebrose, $C_6H_{12}O_6$, is a sugar characterised by its crystallisation, its optical power (its specific or limited rotation being $+ 70° 40'$) and its reducing power over cupro-potassic tartrate. Its theory is the following:—

	Theory		Found.
Of atoms.		Of per cents.	Per cents.
6 C	72	40·000	39·93
12 H	12	6·666	6·71
6 O	96	53·334	53·36
	180	100·000	100·00

Under certain conditions which have been described above cerebrose is changed into an acid isomeric with cerebrose, and therefore of the formula $C_6H_{12}O_6$. This *cerebrosic acid* is dibasic, *i.e.*, contains two atoms of hydrogen replaceable by metals. The theory of the *cerebrosate of barium* is the following:—

	Theory		Found.
	Of Atoms.	Of per cents.	Per cents.
6 C	72	22·85	24·53
10 H	10	3·17	3·2
1 Ba	137	43·49	43·5
6 O	96	30·49	
	315		

This acid is remarkable in this, that it has not got the reducing power over potassio-cupric tartrate, but on the other hand gives with an oleocholide radicle, *e.g.* sphingosin, and oil of vitriol the purple reaction in the same manner as cerebrose.

Psychosin, $C_{23}H_{45}NO_7$ is now fully proved to be the cerebroside of sphingosin; it is crystallisable from alcohol; it is an alkaloid, but of less pronounced character than sphingosin; it forms salts with acids, which are more or less soluble in water, the hydrochlorate being very soluble indeed. By this solubility in cold water it can be separated almost completely from the hydrochlorate of sphingosin, which crystallises from cold water, or cold solution of psychosin hydrochlorate.

	Theory		Found. Per cents.	
	Of Atoms.	Of per cents.	*a.*	*b.*
23 C	276	61·74	61·86	61·32
45 H	45	10·06	10·09	10·09
1 N	14	3·13	2·88	3·04
7 O	112	25·07	25·17	25·55
	447	100·00		

100 parts of psychosin on chemolysis should take up 4·02 parts of water (one molecle), and then split up into 40·29 parts of cerebrose and 63·75 parts of sphingosin.

The hydrochlorate is completely precipitated by excess of hydrochloric acid. Many compounds may be expected to be obtained, as psychosin exhibits numerous promising reactions. It gives the oleocholide reaction with oil of vitriol alone, showing that it contains the cerebrose and sphingosin radicles.

In one chemolysis of phrenosin a body was obtained which I termed *æsthesin*, and found to possess nearly the composition represented by the formula $C_{35}H_{69}NO_3$. The low quantity of oxygen showed at once that it no longer contained the cerebrose radicle. It remained, therefore, to consider it as a compound of sphingosin with neurostearic acid minus water, formed according to the equation:—

$$C_{17}H_{35}NO_2 + C_{18}H_{36}O_2 = C_{35}H_{69}NO_3 + H_2O:$$

	Theory		Found.	Theory of a Hydrate.		
	Of Atoms.	Per cents.	Per cents.	Atoms.		Per cents.
35 C	420	76·22	74·83	35 C	420	73·81
69 H	69	12·52	12·73	71 H	71	12·47
1 N	14	2·72	2·35	1 N	14	2·46
3 O	48	8·54	10·09	4 O	64	11·26
	551	100·00			569	

The æsthesine analysed did, therefore, still contain about half a molecle of water. The body seems to be a base; it is not yet feasible to produce it at will.

The caramels of the nitrogenised bodies described in the chapter relating to these bodies are, like the caramels of the sugars, produced by the expulsion of water under the influence of heat. They are all soluble in ether, insoluble in alcohol and in water, and of a deep brown colour. The following formulæ are hypothetical and interimistic, although derived from the data of the experiments given above.

Caramel of phrenosin, $C_{41}H_{71}NO_4$, formed from a molecle of phrenosin, $C_{41}H_{79}NO_8$, by the loss of four molecles of H_2O.

Caramel of psychosin, $C_{33}H_{37}NO_3$, formed from a molecle of psychosin, $C_{33}H_{45}NO_7$, by the loss of four molecles of water.

A small quantity of caramel is formed during every chemolysis of any of the nitrogenised principles, with acid or with alkali, even during the chemolysis of psychosin with dilute sulphuric acid. There may be several varieties of caramel of each principle produced by the loss of one, two, three or four molecles of water.

Cerebrinic acid has not yet been obtained in a state of purity, but the preliminary tests and analyses show that it contains the same radicles as phrenosin, namely, cerebrose, sphingosin, and neurostearic acid, but in different proportions. The data, particularly the low amount of nitrogen contained in it, lead to the hypothesis that it is cerebrose in which three molecles of hydroxyl are replaced, one by the radicle of sphingosin, and two by two radicles of neurostearic acid, as shown by the equation:

$$C_6H_{12}O_6 + C_{17}H_{35}NO_2 + 2(C_{18}H_{36}O_2) = C_{59}H_{113}NO_9 + 3(H_2O).$$

Cerebrinic acid, as well as kerasin, still require further investigation before their formula can be stated with such absolute certainty as that of phrenosin. It is, however, already almost certain that they contain no saccharide and no nitrogenised radicles other than those contained in phrenosin. There might be mixed with some preparations of phrenosin, particular varieties of phrenosin, in very small quantity, containing in place of the neurostearic, another fatty acid radicle. And there might be mixed with the di-neurostearyl-sphingosyl-cerebroside, or cerebrinic acid, a similarly constituted body containing in place of one or both molecles of neurostearyl other fatty acid radicles. In this respect the nitrogenised principles might imitate the phosphorised principles, which derive their differences from the different fatty acid radicles which they contain, and not from either the phosphorised or nitrogenised radicles, which seem to be the same in all, namely, neurin and glycerophosphoric acid. I think it even very probable that some of the phosphorised bodies which cling so pertinaciously to the nitrogenised ones owe their similarity to these latter, both in shape and chemical properties, to the fact of their containing one or other or both of the radicles of the nitrogenised bodies which appear as neurostearic acid and sphingosin. I state this merely as an hypothesis, worthy of being made the starting point of inquiry. On the other hand, all I have stated as regards the composition and cleavage products of phrenosin may be considered as definite. I have not been able to discover a single datum which would tend to invalidate any portion of the evidence which I have produced on that important subject.

XI.—Note and Experiments on the alleged Existence in the Brain of a body termed Protagon.

Some authors have lately revived what could have been called at one time the hypothesis of "protagon," and have reasserted the existence of this matter as a definite chemical entity or immediate principle in the brain. The result of some new analyses is an empirical formula, $C_{160}H_{308}N_5PO_{35}$, which differs from the earlier formula $C_{116}H_{241}N_4PO_{22}$ by a quantity of no less than $C_{54}H_{67}NO_{13}$. In these analyses also much less hydrogen in proportion to the carbon was found than in the earlier ones, although they refer to virtually the same substance. The processes by which the operators on protagon obtained their matter are essentially the same as those which were employed by Vauquelin, Couerbe, Frémy, Gobley, Bibra, Thomson, and others for the extraction

from brain of a matter which had the same percentic composition as "protagon" with regard to all elements, phosphorus alone excepted, as is evident from the following comparison of the analytical data.

	Frémy's cerebric acid.	Gobley's cerebrin.	Bibra's cerebric acid.	Thomson's cerebric acid.	Liebreich's protagon.	Gamgee and another's product.
C	66·7	66·85	66·78	67·04	66·74	66·39
H	10·6	10·82	10·65	10·85	11·74	10·96
N	2·3	2·29	2·57	2·24	2·80	2·39
P	0·9	0·43	0·52	0·46	1·23	1·06
O	19·5	19·61	19·52	19·41	17·49	19·47

"Protagon" was therefore by no means a needful discovery; and if this substance were really a definite educt it would be absolutely the same as Frémy's cerebric acid. This fact was somewhat obscured by the circumstance that authors on "protagon" ignored the existence of previous researches, and do not even mention the name of Frémy.

Frémy had shown that the body to which he gave the name of cerebric acid was often combined with phosphate of lime, or with soda, and had to be freed from these by treatment with a little sulphuric acid in alcoholic solution. It was this circumstance which induced him to term the body which he had isolated an acid.

Partly in consequence of these observations of Frémy's, I examined all educts from the brain, which I have described in my researches, for mineral ingredients, and found that when they are isolated as far as neutral solvents will isolate them, *they all contain considerable quantities of all, or some, or one of the following bodies: ammonium, sodium, potassium, calcium, iron, copper, calcic, and magnesic phosphates.* (See my "Researches on the Chemical Constitution of the Brain," in the Reports of the Medical Officer, New Series, No. III., 1874, p. 129; also, "Further Researches on the Chemical Constitution of the Brain," No. VIII., 1876, p. 131.) I have given complete quantitative analyses of these bodies as contained in the phosphorised principles discovered by me, namely, *kephalin and myelin.* In one experiment in which the bases were extracted by hydrochloric acid as much as seven grammes of alkaline chlorides were obtained. These inquiries were already, some years ago, extended to the product termed "protagon," and this, too, was found to contain inorganic constituents irremovable by recrystallisation from alcohol, however frequently repeated. It was found more particularly that "protagon" (and the bodies into which it can be separated, according to my researches) always contains considerable quantities of *potassium* in combination.

As the quantity of inorganic ingredients in the "protagon" had never been estimated, I prepared a specimen of this matter, and on analysis found it to contain the better part of one per cent. of inorganic incombustible matters, phosphoric acid not included, and in this no less than 0·76 *per cent. of the "protagon" of potassium.* This same "protagon" contained 1·057 % of phosphorus, and ascribing to it (hypothetically) the mean amount of carbon, hydrogen, and nitrogen found by the latest operators, it would be necessary to consider and rename "protagon" as the potassium salt (mainly) of an acid (in justice to Frémy to be termed cerebric acid), and to give it the formula no longer empirical, but compulsory, if "protagon" were a definite unitary educt, of $C_{284}H_{548}N_9P_2KO_{60}$. If this hypothesis should fail to account for the data, the following conclusion becomes unavoidable, namely, that it is not the fact that any easy process, such as that described for the isolation of "protagon," yields a product in a state of purity, but

that the "protagon" thus far obtained, apart from the question whether or not it is a mixture of organic compounds, is still very impure. The special test experiment (Exp. I.), alluded to in the foregoing, gave results of permanent interest in the consideration of the general question, which deserve to be here recorded. Twelve ox brains were extracted with spirit of 85 °/₀ strength at 45°. All the extracts were cooled to 0°, and allowed to stand over-night. The deposited white matter was collected and exhausted with ether. It was now subjected to the process termed recrystallisation, and thereby split up into the following eight different products: A. Extraction by five litres of spirit at 45° gave a solution which on standing during 16 hours at 17°, without being cooled at 0°, deposited a white matter, the principal bulk of the "protagon," weighing dry 30·9 grm. From this large volumes of ether extracted nearly one gramme of matter (product No. I. 1) containing 0·52 °/₀ of phosphorus. The remaining 30 grammes of "protagon once recrystallised" (product No. I. 2) contained 0·057 °/₀ of phosphorus and 0·76 °/₀ of potassium.

The spirituous mother-liquor which had deposited the foregoing white matter, yielding products No. I. 1, and No. I. 2, on standing during five days at an average temperature of 17°, deposited a voluminous mass of matter, which when dry weighed 3 grm., and contained 1·13 °/₀ P, and 0·22 °/₀ K (product No. I. 3).

The filtrate from this was now placed in water, kept cold by ice during 18 hours, and deposited a matter (product No. I. 4) which when dry weighed 5 grm., and contained 2·02 °/₀ of P, and 0·39 °/₀ K. The spirituous filtrate from this fourth product gave precipitates with cadmic chloride, platinic chloride, and lead acetate, and after distillation of the spirit and drying left 10 grm. of a yellowish, highly hygroscopic residue containing 2·91 °/₀ P, and traces of K (product No. I. 5).

(B.) The white matter from which five litres of spirit had extracted the mixture of the five several products described in the foregoing was extracted twice more with, on the whole, about four litres of spirit at 45°. The solution deposited, on cooling and standing, 8 grm., dry, of a white matter containing 0·76 °/₀ P, and 1·44 °/₀ K (product No. I. 6).

(C.) A considerable quantity of white matter, which had been dissolved in the first spirit at 45°, was left undissolved by spirit at 45° even on very long digestion. It weighed dry 13 grm., and contained large rhombic crystals, resembling cholesterin, but insoluble in either ether or alcohol, and therefore not being cholesterin. Ether extracted from these 13 grm. of residue, 0·4508 grm. of a matter which contained 0·41 °/₀ P (product No. I. 7). The remainder of the matter insoluble in alcohol at 45° and in ether, weighing more than 12 grm., is product No. I. 8.

The following list shows the quantities of the eight products obtained, and their percentages of P and K:—

Numbers of products.	Quantity in grammes.	Phosphorus, per-cent.	Potassium, per-cent.
No. I. 1.	1·	0·52	?
No. I. 2.	30·	1·057	0·76
No. I. 3.	3·	1·13	0·22
No. I. 4.	5·	2·02	0·39
No. I. 5.	10·	2·91	traces.
No. I. 6.	8·	0·76	1·44
No. I. 7.	0·4508	0·41	—
No. I. 8.	12·	—	—
Do. crystallised	10·	0·108	traces.

Here it must be pointed out that all writers on "protagon" have left these products all but No. I. 2 entirely out of consideration, just as they have ignored a large quantity of white matter which is not extracted at all from brain-pulp by spirit at 45°, but requires boiling for solution.

The so-called "protagon" forms only 30 parts out of nearly 70 parts of what remained of the white matter extracted by spirit at 45°, from 12 ox brains after exhaustion with large volumes of ether to such a degree that two litres of ether digested with the matter during six hours left only one decigramme of residue.

Attention may be directed to the fact that in the different products, phosphorus and potassium do apparently observe an opposite ratio, potassium sinking when phosphorus rises, and phosphorus sinking when potassium rises in quantity. The further investigation of these relations will no doubt lead to important and interesting results, but they are foreign to the present purpose.

(D.) *Separation of "protagon" into bodies differing in composition by absolute alcohol at* 45°. The product No. I. 2 above described, corresponding to "protagon once recrystallised," and containing 0·76 % of potassium, was now *treated with* 1¼ *litres absolute alcohol* during 20 hours at 45°. There remained 7·6 grm. undissolved, and this (product No. I. 9) contained 0·96 % P and 1·67 % K. The deposit from the solution on cooling during three hours at 20°, corresponding to "protagon twice recrystallised," weighed 8 grm., (product No. I. 10) and contained 0·83 % P and 1·41 % K. The solution filtered from this, on standing during three days at a mean temperature of 21°, made a further deposit which weighed 4·3 grm. (product No. I. 11), and contained 0·26 % P and 0·22 % K. The mother-liquor on distillation yielded 5·9 grm. of matter (product No. I. 12) containing 1·77 % P and 0·14 % K.

Synopsis of the Products obtained from "Protagon" by Resolution in Absolute Alcohol at 45°, *&c.*

Numbers of products.	Quantity in grammes.	Phosphorus, per-cent.	Potassium, per-cent.
No. I. 9.	7·6	0·94	1·67
No. I. 10.	8·	0·83	1·41
No. I. 11.	4·3	0·26	0·22
No. I. 12.	5·9	1·77	0·14

The matter insoluble in alcohol at 45°, weighing more than 12 grm., (product No. I. 8) was found to dissolve entirely in 300 c.c. of boiling spirit, leaving albumen and paper-fibre, together 1 grm., undissolved. On cooling a deposit rapidly separated, which weighed 10 grm., and contained 0·108 % P, and mere traces of K.

All these twelve deposits contained the usual phosphorised and cerebrin bodies, namely, myelin, phrenosin, kerasin; the mother-liquors deposited kerasin like all other mother-liquors of the cerebrin bodies, (including the boiled ones, as we shall presently show), and gave copious myelin precipitates with cadmic and platinic chloride.

In another experiment (Exp. II.) six ox brains were extracted with large volumes of spirit of 85 % strength at 45°. The solution was allowed to stand at 16°, but was not cooled to 0°. The white matter was isolated and exhausted with ether. It weighed, dry, 25 grm., and contained 0·87 % P. The whole of this *was now boiled in spirit of* 85 %, in which it was completely soluble. It was again deposited on cooling, and the deposit now contained 0·84 % P. The P had therefore been diminished by one recrystallisation. This matter was now differentiated into four fractions by treatment with spirit of 85 % at 45°.

(A.) 2 litres of spirit digested with the "protagon" gave on cooling a deposit (product No. II. 1) which weighed, dry, 11 grm., and contained 0·91 °/₀ P and 0·88 °/₀ K. The mother-liquor left 2·3 grm. of yellowish-white matter (product No. II. 2), which contained 1·83 °/₀ P and 0·87 °/₀ K.

(B.) 1 litre of spirit was digested with the matter which spirit in (A) had left undissolved, for 18 hours, at 45°, and gave on cooling a deposit weighing 3·9 grm. (product No. II. 3) containing 0·74 °/₀ P and 1·04 °/₀ K.

(C.) The matter left undissolved by spirit in (B) was again digested at 45°, with 1 litre of spirit, for six hours. On cooling the solution gave a deposit weighing 1·6 grm. (product No. II. 4) containing 0·46 °/₀ P and 1·23 °/₀ K.

(D.) There were left undissolved by these three extractions with spirit at 45°, 4·4 grm. of matter (product No. II. 5) which contained 0·12 °/₀ P and 0·54 °/₀ K. The mother-liquors of (B) and (C) contained very little matter.

All the fractions summed up, including parts used for analysis, amount to 23 grm., out of 25 grm. originally operated on, so that the amount left in the mother-liquors and lost in operations could not amount to more than 2 grm.

Synopsis of the Fractions obtained in Experiment II. by extracting "Protagon" with Spirit at 45°.

Numbers of fractions.	Quantity in grammes.	Phosphorus, per-cent.	Potassium per-cent.
No. II. 1.	11·	0·91	0·88
No. II. 2.	2·3	1·83	0·87
No. II. 3.	3·9	0·74	1·04
No. II. 4.	1·6	0·46	1·23
No. II. 5.	4·4	0·12	0·54

Here we observe again, as in Exp. I., the peculiarly shifting proportions between phosphorus and potassium. Not a single fraction is free from potassium, and no two fractions contain the same amount of either phosphorus or potassium. All the fractions are mixtures of the same kind, as the fractions obtained in Exp. I., at the same stages; in the corresponding fractions of both experiments P and K observe the same relation, and are present in almost the same quantity; each element at all events observes the same curve in both experiments. The boiling in spirit had therefore not effected any greater difference than the fractional solutions at 45° and precipitations in Exp. I. The appearance of the fractions was the same as that of the corresponding fractions from Exp. I., and on further testing and differentiation, they yielded the well-known constituents just as the products from Exp. I.

In a third experiment (Exp. III.) a quantity of white matter from the human subject was exhausted with ether, and the product, about 60 grm., digested with 4 litres of spirit at 45° for 18 hours. There was left undissolved 4·5 grm. of matter (product No. III. 1.) which after solution in boiling spirit and filtration from some albumin, contained 0·46 °/₀ P and 0·26 K. The solution was cooled rapidly to 21°, and allowed to deposit during 4 hours; the deposit, collected dried, weighed 32 grm. (product No. III. 2), and contained 0·87 °/₀ P, and 0·41 °/₀ K. The filtrate for this principal precipitate was now placed into ice-cold water for 18 hours, and gave a deposit which after isolation was white and wavy, and weighed 8·6 grm. (product No. II. 3); it contained 1·69 °/₀ P and 0·17 °/₀ K. The mother-liquor on

distillation left nearly 12 grm. of a yellowish wavy, hygroscopic body (product No. III. 4) containing 2·85 °/₀ P and 0·09 °/₀ K.

Synopsis of the Fractions obtained by extracting Human "Protagon" with Spirit at 45°.

Numbers of fractions.	Quantities in grammes.	Phosphorus, per-cent.	Potassium, per-cent.
No. III. 1.	4·5	0·46	0·26
No. III. 2.	32·0	0·87	0·41
No. III. 3.	8·6	1·69	0·17
No. III. 4.	12·0	2·85	0·09

The state of aggregation of the particles of "protagon" has been described as crystallised, mostly in microscopic rosettes, but it is admitted that they differ somewhat according to the degree of concentration of the solution from which they are deposited. I inspected microscopically the forms obtained by the recrystallisation of the product No. I. 2, which contained 1·05 °/₀ of phosphorus and 0·76 °/₀ of potassium. I at once perceived the white rosettes of phrenosin, the opaque balls with radiary arrangement of needles of cerebrinic acid, the yellowish balls with concentric layers of myelin, the amorphous particles of another myelin, and over all the long wavy filaments of the blue iridescent kerasin.

The bodies named can be separated out of the "protagon" by the processes which I have described above, I.; the myelins mainly by combination with platinic chloride, or cadmic chloride, or sulphide, or lead oxide. The myelins are not more soluble in ether than the cerebrins, and thereby differ and are easily separated from the lecithins, which are easily soluble in cold ether and cold alcohol, or even spirit; the lecithin platinic chloride is soluble in ether, while myelin platinic chloride is insoluble in ether.

The cerebrins are obtained free from phosphorus mainly by very frequent resolution in hot absolute alcohol. They cannot be separated from each other by this process alone. Lead acetate and a little ammonia are required to make cerebrinic acid insoluble in hot spirit. Phrenosin and kerasin do not permanently combine with lead; they are separated from each other by fractional recrystallisation from suitable volumes of absolute alcohol; phrenosin is deposited earlier at a temperature above 28°, kerasin later at temperatures below 26°, and on long standing. But even so they cannot be liberated entirely from inorganic matters, particularly potassium, and a trace of calcic phosphate; even the lead acetate treatment above described removes only a part, though the greater part of these bases, as acetates. The last traces have to be extracted by cautious treatment with mineral acid in alcoholic solution.

The foregoing data enable us to attribute their proper value to the series of operations by which the advocates of "protagon" have brought about the concordance of their analyses. The potassium, which, though present in such quantity that if "protagon" were a unitary body its atomic weight would thereby be fixed, they have not found, is calculated as oxygen; the phosphate of lime which they have not extracted is made to increase the protagonal phosphorus; the mixture of the myelins which they have not extracted and which they are unable to diagnose is adjusted by solvents to a convenient quantity and made to represent the constituent phosphorus of "protagon"; the cerebrins are not separated from each other, and by a process called slow crystallisation are made to deposit in such a manner that they must be covered over by the only one amongst them which is distinctly

crystalline, and is the one, moreover, which is present in the smallest quantity.

The uniform chemical composition of the brain greatly favours the obtaining from it of extracts of uniform composition; this uniformity can be greatly aided by limitations of the quantities of materials operated upon, and of the quantities and strength of the solvents, and by careful observance of these limitations, preparations are obtained which present a delusive appearance of definiteness.

It follows from the foregoing that the doctrine of "protagon" has been properly rejected by all physiological chemists.

After the publication of the substance of the foregoing paper, the question was again brought under the notice of the Royal Society by Dr. Arthur Gamgee, ("Proceedings of the Royal Society," No. 200, p. 111), and a review of the subject was thereupon laid before the Royal Society by me in a paper entitled "On the Modifications of the Spectrum of "Potassium which are effected by the presence of Phosphoric Acid, and "on the Inorganic Bases and Salts which are found in combination with "Educts of the Brain." This paper, which was communicated to the Royal Society by Mr. John Simon, C.B., F.R.S., etc., and published in the "Proceedings R.S." No. 202, p. 278, Vol. 30, 1880, was as follows:—

Quotation from Proceedings Royal Society.

"Among the results of a large investigation on which I have for many years been engaged in regard of the chemistry of the brain, I had been led to conclude that the so-called 'protagon' of Oscar Liebreich is not a definite chemical body, but is a variable mixture of several bodies. This conclusion of mine (which agrees with opinions expressed on the same subject by Strecker, Diaconow, and Hoppe-Seyler) was published by me in 1874,* and endeavours to controvert it have since then been made, on several occasions, by Dr. Arthur Gamgee.† Last summer, he brought before the Royal Society‡ his contentions for the chemical individuality of 'protagon'; and it fortunately was in my power shortly afterwards to publish evidence, which, I believe, those who will take the trouble to follow it will find quite unanswerable, that Dr. Gamgee's contentions were mistaken.§ Part of my evidence to that effect consisted in showing, by quantitative analyses, that Dr. Gamgee's so-called 'protagon' contains 0·7 per cent. of potassium; secondly, that in connexion with trifling differences in the extraction process, the proportion of potassium in different specimens of 'protagon' can be made to range from a trace to 1·6 per cent.; thirdly, that with the variable quantities of potassium the quantities of phosphorus and other ingredients will also vary.

"In the last published number, No. 200, p. 111, of the 'Proceedings of the Royal Society,' I find that Dr. Gamgee has recently brought the question again under notice of the Society, and that in doing so he especially rests his case upon the following statement made by his colleague, Professor Roscoe, on the subject of some examinations, which, at Dr. Gamgee's request, he had made for him: see 'Proceedings,' vol. xxx., p. 113:—'I have examined spectroscopically for

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* "Reports of the Medical Officer of the Privy Council and Local Government Board." New Series. No. III. (1874).

† "Zeitschrift für Physiol. Chemie," vol. iii, p. 260; "Ber. Deutsch. Chem. Ges.," 1879, &c.

‡ "Proc. Roy. Soc.," vol. xxix, p. 151.

§ See preceding paper.

Quotation from Proceedings of Royal Society.

'potash a sample of protagon furnished me by Dr. Gamgee, and 'labelled 'Protagon, twice recrystallised, Blankenhorn.' I could not 'detect any potash by the spectroscope in the incinerated mass from '0·1 grm. of substance. With the carbonised mass obtained from '1·0 grm. of substance I obtained the potassium line (α) very faintly, 'and from comparative experiments with a dilute solution of a potas-'sium salt I estimate the quantity of potash in 1 grm. of the substance 'not to exceed $\frac{1}{2}$ mgrm. The carbonised residue of 1 grm. of pro-'tagon was carefully oxidised with pure nitric acid, when a small 'quantity of fused metaphosphoric acid remained after ignition. The 'residue weighed 0·0278 grm., corresponding to 1·08 per cent. of 'phosphorus.—(Signed) H. E. Roscoe.'

"As regards the bearing of Professor Roscoe's evidence on the question that has been raised before the Royal Society, and as regards some other relations of the same question, I beg leave to submit to the Royal Society the following observations:—

"1. *On the Modifications of the Spectrum of Potassium, which are effected by Phosphoric Acid.*

"It is well known in spectroscopy that the best spectra of metals are obtained in the easiest manner by aid of their most volatile compounds, such as chlorides; and, on the contrary, that salts which are more or less fixed at high temperatures, such as the silicates and phosphates of alkalies and earths, give either no spectra at all or only very feeble spectra. Thus, if a minute particle of *potassic bromide* be ignited in the aërated gas flame before the slit of the spectroscope, the spectrum of potassium is at once produced in the most brilliant manner: particularly the line in red is brilliant and sharply defined, while the gas flame to the naked eye shows the violet colouring of potassium. But when a similar particle of *potassic phosphate* is ignited under similar conditions, the flame, instead of showing the violet colour of potassium, shows a greenish-yellow colour produced by volatilised phosphoric acid: and in the spectroscope, at first, only the continuous spectrum of phosphoric acid (not to be confounded with the continuous spectrum of potassium) is seen, till gradually, and at a white heat, a feeble potassium line in red, with indistinct edges, is perceived.

"Even a large bead of pure potassic phosphate, when ignited before the slit of the spectroscope, never produces, even at a white heat, any such intense red potassium line as the smallest bead of potassic chloride, potassic bromide, or potassic nitrate. And, in contrast with the latter salts, which volatilise quickly, the phosphate takes some time before it is completely volatilised.

"When such an indolent bead of potassic phosphate which has given a feeble potassium line in red, is dipped into syrupy phosphoric acid and again ignited, the continuous spectrum of volatilising phosphoric acid is again at first obtained, the aerated gas flame to the naked eye is enveloped in a greenish-yellow mantle, and in the spectroscope the continuous spectrum of phosphoric acid only, stretching far into the blue, and without a trace of the potassium line in red, is seen. Gradually the bead flows again as a transparent glass, and then the red potassium line reappears in the spectroscope, though without ever reaching the vivacity of the line produced by volatile salts. If now the bead have added to it a particle of a chloride—for example, zinc chloride—and be again ignited, a lively potassium spectrum will be seen in the spectroscope, *i.e.*, the potassium goes away as chloride. That double decomposition with the separation of the products takes

little more time than the volatilisation of a bead of potassic chloride about the same size.

"Evidently then, if it were intended to estimate from the spectroscopic signs the concentration of a given potassic solution, it would be necessary to take into account not only the intensity of the potassium line produced during the volatilisation of the solution, but also the length of time during which that line is perceived. If solutions equally concentrated of potassic chloride and potassic phosphate are similarly examined before the spectroscope, the phosphatic solution is found to give the feebler band, but to give it for the longest time: and no spectroscopic observation with regard to such a solution can pretend to any validity for purposes of quantitative comparison unless the observer be equally attentive to the duration as to the intensity of the phenomenon.

"Referring now, in the light of these considerations, to Professor Roscoe's letter, I wish first of all to point out that his observation, when made with the carbonised mass from 1 grm. of substance corroborated my assertion that the substance contains potassium. And I may add, by the way, that in my paper on which Dr. Gamgee comments to the Royal Society, I have given 16 quantitative analyses of 'protagon' and its congeners with regard to the quantity of potassium contained in each; having made each determination of potassium by ordinary gravimetric methods, and having weighed the potassium as platinochloride.

"Secondly, however, Professor Roscoe estimates that the quantity of potash contained in a gram of Dr. Gamgee's 'protagon twice recrystallised' did not exceed $\frac{1}{10}$ mgrm., an estimate which he appears to found on the faintness of the potassium line (a) obtained by him from that substance as compared 'with a dilute solution of a potassium salt.' This inference I think it necessary to check by the considerations I have before stated. Professor Roscoe states that he obtained from 1 grm. of 'protagon' a residue of 0·0278 grm. of metaphosphoric acid, but according to his spectroscopic estimate, less than $\frac{1}{10}$ mgrm. of potassium. What is the value of the latter half of this statement when taken with the context of the other half?

"Before Professor Roscoe could possibly obtain from such a substance as he seems to have examined any spectroscopic evidence of the presence of potassium, he would have had to drive away from his platinum wire a relatively enormous amount of free phosphoric acid. He would then have had some potassic phosphate left, which, as we have seen, gives only a feeble and indistinct potassium band in the red, even when a large bead is heated in the flame before the spectroscope, and which, in so extremely small an amount as he must have had, would of necessity give a fallaciously weak reaction.

"Further Professor Roscoe seems not only to have thus overlooked the inhibitive or weakening influence of phosphoric acid in relation to the spectrum of potassium, but has also omitted to give any objective measures of the intensities which he purports to compare, has moreover not recorded the length of time during which the compared phenomena continued, and has not even named what was the particular potassic salt used by him as a standard in his comparison.

"Apart, therefore, from the inherently small worth of spectroscopy as a quantitative method, not even those precautions of which the spectroscopic method admitted were employed by Professor Roscoe, and I must therefore respectfully submit my opinion that his experiment is of no practical value."

App. B. No. 3.

On the Chemical Constitution of the Brain and of the Organoplastic Substances, by Dr. Thudichum.

Quotation from Proceedings of Royal Society.

"2.—*On the Inorganic Bases and Salts which are found in combination with the Educts of the Brain.*

"Frémy, in his research on the brain published in 1841, stated that the body which he had isolated and termed 'cerebric acid,' was frequently combined with soda and phosphate of lime. Another substance which he termed 'oleophosphoric acid,' he assumed to be ordinarily in the state of soda salt, but sometimes combined in part with phosphate of lime. He separated these bases and salts by dissolving the mixture of the two bodies (cerebric and oleophosphoric acid) in boiling absolute alcohol, which had been made slightly acid by sulphuric acid. Sulphates of lime and soda remained suspended mixed with some insoluble organic matter, and were removed by filtration. The cerebric and oleophosphoric acids were in solution, and deposited on cooling. From the mixture oleophosphoric acid was extracted by ether, which left the cerebric acid behind. Frémy further found that cerebric acid combined with all bases, and must be considered a true acid. When heated with dilute solutions of potash, soda, or ammonia it did not dissolve, but combined with each of these bases. The compounds were also obtained by mixing any of these bases with an alcoholic solution of cerebric acid. Thus, he prepared the insoluble barium compound containing 7·8 per cent. of baryta.

"Bearing this experience of Frémy in mind during my researches on the chemical constitution of the brain, I invariably examined for inorganic ingredients all the educts which I obtained from the brain.*

"Of KEPHALIN (the most important of the phosphorised ingredients of the brain) 10 grms. were dissolved in one litre of water, and after filtration precipitated by hydrochloric acid. The solution of hydrochloric acid and other matters filtered from the precipitated kephalin was evaporated to dryness. A portion was then boiled with solution of baryta, when traces of *ammonia* were evolved. The rest of the residue was then ignited in a platinum dish to destroy all traces of organic matter. The ash was but slightly fusible; was only partially soluble in water, but was easily soluble in water slightly acidified by hydrochloric acid. The solution with ammonia gave an abundant precipitate of *earthy salts*, and the solution assumed a *deep blue colour*. Precipitate and solution were separated by filtration.

"The precipitate dissolved readily in a little hydrochloric acid, forming a slightly red solution, suggesting the presence of *iron*, which was confirmed by the sulphocyanide test. In another portion of the solution dilute sulphuric acid gave an abundant precipitate of gypsum showing presence of *calcium*. In another portion the molybdate test showed the presence of *phosphoric acid*. To the remaining portion a few drops of ferric chloride were added, then sodic carbonate nearly to neutrality, and lastly, barytic carbonate. The mixture was allowed to stand, filtered, and the filtrate, free from excess of baryta by sulphuric acid was again filtered, and the filtrate, after supersaturation with ammonia, gave a great precipitate with ammonic oxalate, showing presence of much *calcium*. The filtrate from this calcic oxalate on concentration, and treatment with ammonia, ammonic chloride, aud sodic phosphate, gave the precipitate characteristic of *magnesium*. The precipitate produced as above described by baryta carbonate, was

* That part of my experience relating to kephalin and myelin is recorded in "Reports of the Medical Officer of the Privy Council and Local Government Board," new series, No. III (1874), p. 129, and *ibid.* No. VIII (1876), p. 131.

boiled with excess of pure soda, and the filtrate was warmed with ammonic chloride; only a slight turbidity ensued, so that it may be assumed that *aluminium* was absent.

"The *alkaline filtrate* from the foregoing precipitate by ammonia was tested for lime by oxalate, when a considerable precipitate was produced, showing the presence of *lime uncombined with phosphoric acid*, and which therefore must have been in combination with part of the kephalin. The blue solution was again filtered from calcic oxalate, which had been entirely precipitated, and was acidified with hydrochloric acid. The *copper* was precipitated by hydrothion, the filtrate evaporated to dryness, ignited, and the residue tested for alkalies. This residue exceeded in bulk the bases previously removed. It was fusible with ease, and on solidification became white and crystalline, but interspersed with many red particles of ferric oxide. It was a mixture of *potassic and sodic chlorides*, and of some *ferric oxide*, which, owing to the presence of the chlorides, had escaped precipitation by excess of ammonia.

"It was, therefore, proved that the kephalin, obtained by the alcohol and ether process, and purified by solution in water and filtration, as described at length in my researches above quoted, is, at least in part, combined with *ammonium, sodium potassium, calcium, iron, copper*, and *calcic and magnesic phosphates*. This experience was repeated a great number of times, and in the aggregate on several hundred grams of dry kephalin.

"In the following experiments MYELIN (the phosphorised ingredient which stands next in importance to kephalin in the chemistry of the brain)* was examined for inorganic bases, in order to obtain some knowledge regarding their relative amounts and nature. The myelin was dissolved in water, precipitated by hydrochloric acid, and the acid solution separated by filtration. The *copper* was removed by hydrothion, the filtrate evaporated to dryness, and the residue ignited in a platinum dish. During this ignition much *ammonic chloride* was given out, and its nature distinctly proved by condensing and analysing it.

"The fused mass was dissolved in dilute hydrochloric acid, and the solution so obtained treated with excess of ammonia. The white gelatinous precipitate which was thrown down was scarcely coloured black by ammonic sulphide, indicating the presence of only a trace of iron. The precipitate and filtrate were submitted to analysis, qualitative and quantitative, as follows:—

"*The precipitate* was dissolved in dilute hydrochloric acid, the solution was nearly neutralised by sodic carbonate, after addition of some ferric chloride; an excess of barytic carbonate was now added, the mixture was shaken, allowed to stand, and then filtered. From the concentrated filtrate the barium was then removed by sulphuric acid, and the filtrate was precipitated in the presence of excess of ammonia by ammonic oxalate. The oxalate so obtained was converted by intense ignition into oxide, which weighed 0·1994 grm., equal to 0·1423 grm. *calcium*. The filtrate and washings from the calcic oxalate were treated as usual for the quantation of magnesiun; and there was obtained 0·5556 grm. magnesic pyrophosphate, equal to 0·1212 grm. *magnesium*. The precipitate which had been produced by ferric chloride, and sodic and barytic carbonates, was dissolved in nitric acid, and the phosphorus estimated by precipitation

* Reports, as above, No. III, p. 156.

with ammonic molybdate, and transformation of the precipitate into magnesio-ammmonic phosphate, &c. This gave 0·3266 grm. *phosphorus*, equal to 0·7480 grm. phosphoric anhydride.

"*The filtrate* was proved to be free from magnesium and calcium. The potassium contained in it was extracted in the usual manner, by means of platinic chloride, giving 11·2766 grms. of the double salt ($2KCl,PtCl_4$) equal to 3·651 grms. potassic chloride, or 1·911 grm. of *potassium*. The mother liquor and washings were evaporated to dryness, and the platinum was removed by extracting the ignited mass with water acidified by hydrochloric acid. The extract so obtained was evaporated to dryness and fused; the residue weighed 3·45 grms., being pure sodic chloride equal to 1·356 grm. sodium. The quantities of sodium and potassium thus found were controlled by the analysis of a separate part of the original solution containing their chlorides; and on calculating from the residue obtained on evaporation there should have been found a total quantity of chlorides = 7·09 grms. There was found potassic chloride = 3·651, and sodic chloride = 3·450; together, 7·101 grms.

"Assuming the above earthy bases to have originally existed in myelin as tribasic phosphates, and any phosphoric acid remaining over to have been in combination with potassium, then we have,

Calcium	- = 0·1423 grm.	existing as	$3CaO,P_2O_5$.
Magnesium	- = 0·1212	„ „	$3MgO,P_2O_5$.
Potassium	- = 0·5663	„ „	$3K_2O,P_2O_5$.
Potassium	- = 1·3447	„ combined directly with	
Sodium -	- = 1·3560	„ myelin.	
Phosphorus	- = 0.3266	„ existing as P_2O_5 (0.748 grm.)	

and distributed between the calcium, magnesium, and potassium.

"In the kephalin research, related above, it was proved that kephalin was partly in combination with calcic oxide or lime, without any other acid being united with that base. In the myelin research it was found that, in myelin, there may be in combination even a much greater quantity of potash and soda than of lime.

"A third research was now made on the mixture of phosphorised and nitrogenised substances, which Frémy termed 'Cerebric acid,' and which later writers have termed 'Protagon.' This after exhaustion with ether, was redissolved eight times in hot spirit, and after filtration reprecipitated on cooling. It was pressed to remove all mother-liquor, was then suspended in water and heated until it became thoroughly emulged with it; acid, either sulphuric or hydrochloric, was now added, whereupon the organic matter curdled and contracted; it was separated, and the acid solution was treated like the above described similar solution from kephalin and myelin. Considerable quantities of the same bases and salts as those obtained from kephalin and myelin were isolated, but not examined any further.

"It had been ascertained in previous operations on these mixed phosphorised and cerebrin bodies, and was now again observed, that one treatment with acid was insufficient to free them entirely from bases. The bodies which had been treated with acid were, therefore, treated with lead acetate solution in the cold, and were allowed to stand. The liquid which formed was filtered from, and pressed out of the lead-compounds, and on proper treatment yielded as follows:—*sodium*, = 1·78 grm.; *potassium*, = 1·185 grm.; *calcium*, = 0·08 grm.; and *magnesium*, 0·02 grm. The sodium was weighed as chloride; the potassium, 0·856 grm., as bitartrate, and 0·32 grm. as platinic chloride; the calcium as carbonate: and the magnesium as

pyrophosphate. The respective preparations are herewith submitted for inspection.

"Curious facts which deserve notice are the following. When so-called 'protagon' was decomposed by baryta to obtain neurin, it was observed that the platinic chloride salt of the base always *contained potassium*. When the neurin was combined with phosphomolybdic acid, and carefully washed, and then again transformed into platinic chloride salt, it became free from potassium. In both cases the salt was crystalline, but the crystallisation was different in the two cases. The crystals containing potassium were most perfectly formed individuals, sometimes more than half an inch in length and breadth, and an eighth of an inch in thickness, while, on the other hand, the crystals free from potassium were bundles of needles, of lighter aspect and not admitting of angular measurement. The experience shows that the platinic chloride compound of potassic chloride is dimorphous, and in one of its forms is isomorphous with platinic chloride salt of neurin hydrochlorate. It is further remarkable that the impure isomorphous mixture crystallised in well-defined indviduals, while the pure salt crystallised in confused masses and in bundles of needles. The crystals of the perfect type, when powdered and burnt on a platinum wire in an aërated gas flame before the spectroscope, produced the line in red characteristic of potassium. This specimen is herewith submitted for inspection, as also a specimen of the salt free from potassium.

"The foregoing data may perhaps contribute to explain some of the discrepancies which have been found between different specimens of neurin platinic chloride, and have caused the inconvenience of a number of different formulæ being attributed to neurin, namely, $C_5H_{15}NO_2$, or $C_5H_{14}NO$, or $C_5H_{14}N$, or $C_5H_{12}N$. They may also explain why neurin from 'protagon' has been supposed to consist of a mixture of two bodies, having respectively the formulæ $C_5H_{15}NO_2$, termed oxyneurin, and $C_5H_{15}NO$, termed neurin. According to my own numerous analyses (Reports as above, No. VIII, p. 118, *et seq.*), the principal ammonium base from the phosphorised substances, has, after purification by the phosphomolybdic process and combination with hydrochloric acid and platinic chloride, invariably the composition $C_5H_{13}NO$.

"The stoichiometric relations of such a base, in its platinic chloride salt, would be doubly disturbed by the presence of potassium; for not only would a portion of the platinic chloride, namely that combined with potassic chloride, be erroneously referred to neurin, but the entire amount of platinum, if estimated, as is usual by combustion, would be raised in apparent amount by the presence of the potassic chloride as an invisible impurity.

"The albuminous ingredient of the brain contains considerable amounts of the usual salts, particularly calcic phosphate, of which the calcium is obtained as gypsum, when the albumin is treated with dilute sulphuric acid, and the solution of hemi-albumin is filtered from the undissolved hemi-proteïn and slowly evaporated. By combustion of the albuminous matter of the brain, all the usual salts and bases, including copper, iron, and manganese, are liberated.

"The greater part of the *soluble salts* contained in the brain passes into the water extracts, while the greater part of the *insoluble salts*, such as phosphates of earths, remain with the albumin; but a portion of both remains with the phosphorised and nitrogenised principles, and follows them into all their solutions in ether and alcohol. Only *sulphates*

Quotation from Proceedings of Royal Society.

have not been found in these latter, though they are present in the water extracts.

"It follows that all educts from the brain have to be freed from the salts or bases described before they can be subjected to quantitative chemical examination; and an error which seems to me to pervade all hitherto analyses of the brain for inorganic ingredients is the following. When the brain is burnt in its entirety, the phosphoric acid resulting from the destruction of the phosphorised compounds expels sulphuric, hydrochloric, and carbonic acids, and comes to be considered as an inorganic ingredient, the fact of its previous combination being overlooked. When the brain is freed from matter soluble in spirit and ether, and the albumin and water extract alone are examined for inorganic ingredients, the fallacy otherwise introduced by the phosphoric acid in organic combination is no doubt avoided; but the inorganic salts described above, as in combination with the phosphorised and nitrogenised matters are overlooked and left out of consideration.

"A vast range of other organic analyses (animal and vegetable) for inorganic constituents is no doubt affected by the same fallacy, and it seems to me that the right making of such analyses is a much more complicated problem than has hitherto been believed"

II. Further Researches on the Chemical Constitution of the Organoplastic Substances.

No. I.—The Chemical Constitution of the Organoplastic Substances considered with the aid of the Hypothesis of their Amylonide Nature.

The material which forms the solid and rigid parts of the structure of plants is *Cellulin*. This name has been selected because microscopical science has found that the simplest-shaped elements of vegetable structure are cells, and that all more complicated structures arise out of aggregated cells by processes of peculiar union and fusion; that the rigid woody part of cells is also the principal part when its quantity is compared with that of other ingredients, and that there are no cells discernible made of any other material. The youngest cells, such as those in the first rootlets or leaves of germinating seeds, contain the purest cellulose; with advancing growth and age this becomes covered with new homogeneous layers. Other additions are heterogeneous, and varying in chemical composition, but one kind of matter is observed to be closely associated in some form or other with growing cellulin in all its varying forms and positions, namely, a nitrogenised matter resembling the radicle which has been supposed to be the nucleus of the albuminous substances (Protein). With advancing age and ceasing increase the cells assume a final rigid shape, which they maintain unaltered during the remainder of the existence of the organism of which they form part. The nitrogenised matter at the same time leaves them almost entirely, and they become wood. Not rarely this wood at the time of involution of the cell-life receives an addition in the form of deposit or incrustation of mineral matter, mainly carbonate, and sometimes phosphate of calcium, silica, potash, and other salts.

The chemical composition of cellulin finds its most simple expression in the formula $C_6H_{10}O_5$, but it is supposed that its molecle is a multiple of this formula, and therefore it is expedient to express its formula by $n\ (C_6H_{10}O_5)$. This formula also has the advantage of including the objects of those theories according to which there are

several varieties of cellulin, some being represented as isomers, some as polymers of what might be termed the type.

In the growing parts of plants already developed cellulin is probably produced directly from and in the protoplasm, which, with its nucleus, serves as the centre of nutrition and multiplication, and as the mould, so to say, over which the actual cell of cellulin is formed. The cell is in other words the shell which protoplasm forms on its outside. But in seeds and tubers, and other organs effecting the propagation of the species by whatever means, a material for the formation of cellulin is ready for use deposited inside the cells, namely *starch* or *amylon*. This material is not used in the very cells in which it is deposited, but is only stored there in a form insoluble for the time, but capable of being liquefied and carried to and employed in newly-growing parts at the call of heat and light. Amylon also has the empirical formula of $C_6H_{10}O_5$, but most inquirers agree that this formula has at least to be doubled to express the considerations which arise from the simplest reaction. We are, however, justified in going further, and admitting, though hypothetically, that amylon, like cellulin, is best expressed by the formula $n\ (C_6H_{10}O_5)$ in which n is a factor not less perhaps than five, and in given conditions probably more.

In considering amylon as a chemical individual, it must be borne in mind that it is perhaps not identical with the whole of the product termed starch, but that a portion of the granules constituting this substance may be, as some maintain it is, actually cellulin while another is amylon, and that in the concentric arrangement of the starch granules, layers of these substances alternate with each other. This discussion, however, is immaterial to our present purpose, because the fact remains, however the discussion may be decided, that the whole starch granule is available for fluidification by a ferment, and for transfer to parts newly formed or in course of growth.

In other parts of vegetables we find carbohydrates which closely resemble cellulin and starch, and some of them are formed out of starch, either by natural or by artificial processes. These are the dextrins, sugars and gums. Dextrin from wheat starch has, like this, the composition $n\ (C_6H_{10}O_5)$; gum arabic or arabic acid, however, is at least $C_{12}H_{22}O_{11}$; while bassorin, the gum of tragacanth, remains like dextrin $n\ (C_6H_{10}O_5)$, and vegetable mucilage the same. In dextrin and gums the factor n may possibly be $= 1$, or $= 2$, probably not more. The sugars have a great variety of formulæ, and are many in number. Pinite or pine sugar is $n\ (C_6H_{12}O_5)$; Quercite, the sugar of acorns, has the same formula. Milk sugar or lactin is $C_{12}H_{22}O_{11}$; cane sugar has the same formula. Passing over a number of peculiar saccharoids occurring in rare plants we pause at the glucoses, the one which turns the ray of polarised light to the right being dextro-glucose, $C_6H_{12}O_6$, and the one which turns it to the left, or lævo-glucose, being perhaps of the same formula, but possibly, $C_{12}H_{24}O_{12}$. Closely related to these is maltose, $C_{12}H_{22}O_{11}$, the sugar obtained by the action of diastas from starch. Next we notice mannitose, then mannite, $C_6H_{14}O_6$, a hydrogenised sugar, which can be produced by inserting hydrogen in a sugar with 12 atoms of hydrogen. Next follow some sugars which are common to plants and animals, namely inosite (phaseo-mannite) occurring in the muscles and brain of vertebrates, and dextro-glucose occurring in the blood and parts of animals; and scyllite in organs of the ray, shark, and prickly dogfish.

Lastly, we cast a glance at the glucosides, so termed because by chemolysis they yield glucose and other products. These are present

in many plants under remarkable circumstances. Bodies like them occur in animals, but they are to be regarded as peculiar principles.

The carbohydrates of the order of gums and sugars do not as such serve for purposes of structure, but as vast as are the structure functions of cellulin so are the power-producing functions of the sugars. The sugars are stores of matter or power for the accomplishment of the propagation of the species in plants; roots, rhizomes, bulbs, stalks, leaves, and buds, but, above all, fruit, abound in sugar. Lastly, in the living body of animals there is always sugar formed and present, whatever may be the mode of nutrition. Sugar seems at present incapable of being artificially metamorphosed back into cellulin; neither does the metamorphosis appear to be effected in animals or plants.

The discovery by Schwann in 1840, that the elements of structure were the same in plants and animals, was followed in 1845 by the discovery of K. Schmidt (Zur vergl. Physiologie der wirbellosen Thiere 1845, p. 62; Ann. Pharm. 54, 318: J. Pr. Chem. 38, 433) that some of the lower animals were provided with parts consisting of cellulin. These were the Tunicata; in one species of these, and more particularly in the mantle of Phallusia mamillaris, cellulin—as it was believed to be—was met with. The discovery was extended by Löwig and Kölliker (Ann. de Sc. Nat., 3 Ser. V., pp. 193–232: J. Pr. Chem., 37, 439; Compt. Rend., 22, 38, 1846) who, confirming Schmidt as to the presence of cellulin in Phallusia mamillaris, met with the same constituent in Phallusia intestinalis, Phallusia monachus, Cynthia papillata, Clavelina lepadiformis, Diazona violaceum, Botryllus polycyclus, Pyrosoma giganteum, Salpa maxima, Didemnum candidum, and Aplidium gibbulosum. They found the middle layer of the mantle of Phallusia monachus and sulcata, of Clavelina lepadiformis, and Aplidium gibbulosum to be composed of a glass-like transparent structureless substance in which many nuclei and crystals arranged in starlike groups were imbedded; the external layer of the mantle they observed to be crowded with very large, elegant, round cells with thin walls enclosing a matter clear like water, but no nucleus.

Schmidt also believed he had found an infusorium, Frustulia salina, to contain cellulose. It was however afterwards shown that this creature is not an infusorium but a plant (Siebold, Vergl. Anat. d. wirbellosen Thiere, 1848, p. 238).

In Clavelina lepadiformis the entire mantle on the stalk, as well as the sprouts, is permeated by such roundish and oval cells without nucleus to such a degree that the homogeneous substance in which otherwise they are imbedded has disappeared. Such layers of the mantle have then entirely the appearance of vegetable tissue. In Aplidium gibbulosum and Botryllus violaceus the tender cells situated in the more external layer of the mantle contain calcic carbonate, which increases towards the periphery, so that the cells of the outermost layer appear completely calcified. In Didemnum candidum these calcified cells, which are here moreover provided externally with rays of calcic carbonate, appear in such numbers that the entire stock of this compound ascidia appears to be penetrated by white star-like bodies. Similar to this appears, according to Milne Edwards, (Observ. sur les Ascidies composées, p. 81, Pl. 1, Fig. 2nd), Leptoclinum maculosum. In the mantle of Diazona violaceum, Pyrosoma giganteum, Botryllus polycyclus, Salpa maxima and bicaudata these tender delicate cells are not seen, but the homogeneous substance mainly contains disseminated granules and nuclei, in Pyrosoma also cells with peculiar branches, and in Diazona coloured granules, and needle-shaped crystals or concretions of calcic carbonate. Such crystalline concretions in the shape of balls

or stars are also present in Salpa maxima, and others in branched masses in Salpa bicaudata, but they are not soluble in hydrochloric acid, and are therefore probably not chalk but silicic acid. In Botryllus the homogeneous substance of the mantle is in certain places penetrated by peculiar serpent-like fibres which twist in all directions, and on treatment with caustic potash are recognised to be wood fibres free from nitrogen. In Cynthia papillata the middle layer of the mantle consists of wavy wood fibres arranged longitudinally and perpendicularly, with interspersed nuclei, granules, crystals, and cells; the latter contain either brown pigment granules, and a nucleus, or several endogenous cells, by which they recall the appearance of cartilage cells. The fibres of the middle layer of the mantle aid in the production of the thorny projections which appear on the otherwise horny-looking surface. In the mantle of Cynthia pomaria longitudinal fibres prevail, between which crystals, round pigment cells, and peculiar cells filled with yellow bodies are situated. With these cells are mixed a quite peculiar kind of cells, which are developed from pigment cells and form thick walls during their growth; these walls then break up into fibres, which latter now surround the cavity of the former cell in concentric layers. These fibrillated cell-membranes react with potash as little as the principal substance; they are wood fibre, free from nitrogen; all other elements of the tissue disappear in the caustic alkali. (Siebold, Vergl. Anat., p. 238, Anm. 4.)

No such matter free from nitrogen and resembling cellulin was found by these observers in other molluscs, in annulates, helminths, echinoderms, discophores, polyps, and infusoria.

In publishing their researches they expressed the apprehension that this discovery would be used by those who deny the existence of any precise demarcation between animals and plants for the purpose of supporting the opinion that as regards chemical composition also there was no fixed difference between plants and animals. They therefore exerted themselves to prevent such a hasty conclusion, as it was termed, and pointed out particularly that the woody fibre of the tunicata was never contained in the mantle of these animals in a quite pure state, but was always yet mixed with animal elements, and that there existed no lower animal which consisted in all its parts of cellulose. (Siebold, l.c., p. 238, Anm. 1.)

The researches of Löwig and Kölliker formed the subject of a report to the French Academy by Dumas, Edwards, Boussingault, and Payen, published in the Compt. Rend., 22, 581; Ann. Sc. Nat., 1846, 238.

Thus the matter rested for about 12 years, when Berthelot published some new researches on the peculiar substance of the mantle of Cynthia papillata (Compt. Rend., 47, 227; N. Ann. Chim. Phys., 56, 149, J. Pr. Chem. 76, 371.) As the result of these he maintained that this matter was not identical but only isomeric with cellulin. On chemolysis with sulphuric acid it yielded fermentable sugar, and another matter not examined. He declared it to be rather analogous to chitin, termed it tunicin, and gave it the formula $C_6H_{10}O_5$ or $C_{12}H_{20}O_{10}$.

About the same time Berthelot found that chitin gave by chemolysis a fermentable sugar free from nitrogen. This was at first referred to the presence with chitin of some such body as tunicin, and Peligot (see reference in the subsequent paper), actually endeavoured to find and extract the amylum-like body which was supposed to yield the sugar. It is by no means certain whether such a body does not exist mixed with chitin; but chitin itself is nitrogenous, and therefore not at first sight so like cellulin as it would appear if it were free from nitrogen.

Schmidt, the discoverer of animal cellulin, had studied chitin also, and found that by thermolysis it gave out acetic acid and ammonia. But his observation was not appreciated by Städeler in his research on chitin; he occupied himself with constructing a new formula for the body on the basis of its empirical composition; he did not suspect that the sugar obtained by its chemolysis in a certain way contained nitrogen. That under certain conditions it did contain nitrogen was only discovered in 1876.

The article on chitin on a subsequent page will explain how closely chitin is related to tunicin or animal cellulin, and to the amylonides of the vegetable world which form the permanent structure of plants. It is indeed somewhat more complicated in chemical structure, inasmuch as it contains nitrogen, but even that is present in the rather simple form of amide. Beyond this amide there are some simple radicles present which appear as fatty acids after chemolysis, but the radicle stock determining the nature of chitin is a body like cellulin, an amylonide.

Though Löwig and Kölliker had not found any cellulin in helminths, a body was soon discovered in the peculiar cystic form of the Taenia echinococcus, which resembled in these respects the cartilaginous covering of some of the lower animals, that by chemolysis with sulphuric acid it yielded dextro-glucose. The bladders of echinococci were found by Lücke (Archiv. Patholog., 19, 189 ff.) to be made of a nitrogenous substance, which dissolved in hot water under pressure, like one of the permanent cartilaginous substances. The membranes of old bladders were in part insoluble in caustic alkali even on long boiling.

The fully-developed helminths contain probably no such matter in their cuticle, though they are, like the bladder, covered with calcic carbonate. But they are covered probably with an amylonide, and at all events their parenchyma (intestinal helminths) is pervaded by large quantities of so-called (hepatic) glycogen.

In the mollusca provided with shells, a peculiar substance exists, mainly as the organic base of the shells. This was for some time believed to be identical with chitin, but was at last separated by Frémy, and later by Schlossberger, and distinguished under the name of *Conchiolin*. It is at once distinguished from chitin and hyalin by the amount of nitrogen, which is similar to that of the albuminous substances:—

	Hyalin.	Chitin.	Byssin.	Spongin.	Conchiolin.
C	44·55	45·69	—	47·44	50·0
H	6·64	6·42	—	6·30	5·9
N	4·81	7·00	13·5	16·15	17·5
O	—	—	—	—	26·6

The only crystallisable product as yet obtained from it is *leucin*; this would range it under the animal matters to be considered later on.

The byssus of the acephala was also supposed to contain chitin Leukart), but apart from its reactions, which are peculiar, it contains from 12·2 to 13·9 % of nitrogen, and therefore differs from both chitin and conchiolin. The byssus of the mussel when free from impurity and a little calcic carbonate, is a mass of fibres shining like silk, and elastic like caoutchouc, and seems at first sight to have little physical similarity to other animal substances except elastin. Spongin has yielded leucin and glycin, but not a trace of tyrosin. It has not yet been examined for any amylonide; but as some declare it to be identical with fibroine, which has been found to yield sugar, further examination may furnish information on this point. The nitrogen in spongin is 16·5 %, and this ranges the substance between byssin and conchiolin. Fibroin,

the substance of silk, has yielded on chemolysis tyrosin (in the large quantity of from 5 to 8 %), leucin and other amido-acids, ammonia, sugar, recognisable by an alkaline solution of cupric oxide, and especially, on long boiling, glycin. The composition of fibroin is similar to that of conchiolin, $C_{15}H_{23}N_5O_9$ expressing its empirical formula.

Heated with caustic potash it easily forms oxalic acid. It is well intermediate in chemical structure, between the cellulin on the one, and the proteins on the other extremity of the scale.

Silk gelatin has yielded tyrosin, leucin, and sericin, $C_3H_7NO_3$ (amido-oxy-propionic acid), which yields glyceric acid by treatment with nitric acid. Silk gelatin is the outer covering of the silk fibroin, which forms the inner fibre; its composition is expressed by the empirical formula $C_{15}H_{25}N_5O_8$; it thus appears to contain a molecle of water, and one of oxygen more than fibroin. The difference is, however, greater than that indicated by the one in hydrogen and oxygen, inasmuch as the radicle represented by sericin has not yet been observed amongst the products of the chemolysis of fibroin.

Mucin is the substance characterising the mucous secretions and excretions of a great number of animals. Snails secrete it in large quantities to prevent adhesion to objects in their progress, and they lubricate their way with it. Inversely almost all animals form it in their interior parts to prevent adhesion to them of foreign bodies, like food, or adhesion to each other in parts exposed to friction or the drying effects of the air. It exists in quantity in the glands forming the lubricating materials of the digestive canal, notably the salivary glands, but it is still more singular that the lubrication of tendons in their sheaths should be attained by it, and that the tough materials of the tendons themselves and of all connective tissue whatever should contain a large amount of mucin. Its formula has not yet been attempted; it is separated from the albuminous matters by its insolubility in strong acetic acid. By chemolysis with mineral acids, or even by prolonged boiling with moderately strong acetic acid, it gives a kind of acid albumin and grape-sugar, and probably other substances. The grape-sugar has been isolated and identified with alkaline copper solution, and with potash ley (Eichwald).

The analyses of mucin differ as yet too much from each other to allow the attempt of constructing a formula. Nitrogen is found by some at 8·5, by others at intermediate figures, 9·42, 10·01, 12·64, 13·22, 14·54, 17·69, and not all of these have been shown to form sugar. But enough has been shown to make mucin a factor in the argument here proposed. It is an element of structure, and an agent of function, and from its qualities eminently fitted for both performances.

Mucin is also a product of diseased action, as it occurs in the fluid contents of colloid sacks, in sarcomatous and in fibrous tumours. *Chondrinogen*, or cartilage, is by itself insoluble in water. It constitutes the permanent cartilages, the bone cartilages before ossification, and the cornea of the eye. In growing stag's horn it is contained together with ossein, as it is also present in the bones of young individuals of all vertebrates. Rib-cartilage purified with dilute hydrochloric acid, when boiled with fuming hydrochloric acid, yields fermentable sugar. (G. Fischer and Boedeker, Ann. Pharm., 117, 111: see also Schiff, Ann. Pharm., 119, 256.)

This sugar, which Boedeker had formerly described as chondroitic acid (Schmidt's Jahrbücher, 90, 150), was found by De Bary to be lævo-rotatory, and to differ from both dextro-glucose and lævo-glucose (Chondrose). Chondrin yields by fusion with potash, oxalic acid, leucin, glycin, and an acid which is fixed and gives a crystalline lime

salt. It sometimes forms a jelly with water on cooling like gelatine, therefore like starch. The products of the chemolysis of chondrin under varying conditions are very imperfectly known, but nevertheless it is clearly marked as an amylonide.

Ossein, the organic basis of bones and teeth, is also in part the organic basis of reindeer's feet, stag's horn, and whalebone. In boiling water it is slowly converted into an equal weight of gelatin, more rapidly when prepared from the bones of young animals, and very speedily after addition of small quantities of acid. It contains—

$$C = 50{\cdot}4 \qquad H = 6{\cdot}5 \qquad N = 16{\cdot}9 \qquad O = 26{\cdot}2$$

The bones of fish and of aquatic birds contain, besides ossein, a substance insoluble in boiling water, yet apparently of the same composition as ossein. When these bones are freed from lime-salts by cold dilute hydrochloric acid, and the residue is washed free from acid and then boiled with water, the ossein dissolves as gelatin, while a transparent elastic residue, having the form of the bones, remains undissolved. (Frémy, N. Ann. Chim. Phys., 43, 51; N. J. Pharm. 27, 1.)

Gelatin is also obtained from the gelatinous tissues, the intercellular or so-called connective tissue, from tendons, ligaments, and lining membranes of sacs and cavities in the animal organism.

Elastic tissue and fibro-cartilage produce a somewhat peculiar gelatin.

Already 30 years ago Hunt (Amer. Journ. of Sc., Jan. 1848, p. 74, 109), perceived that when he added the elements of ammonia to those of cellulin or of amylon and then deducted the elements of water, he obtained a product which had very nearly the formula of gelatin.

$$\underset{\text{Cellulin}}{C_6H_{10}O_5} + \underset{+\text{ ammonia}}{2NH_3} = \underset{\text{gelatin}}{C_6H_{10}N_2O_2} + \underset{+\text{ water.}}{3H_2O.}$$

The per-centage composition of a body n ($C_6H_{10}N_2O_2$) would be—

$$\begin{aligned} C &= 50{\cdot}70 \\ H &= 7{\cdot}04 \\ N &= 19{\cdot}71 \\ O &= 22{\cdot}55 \\ \hline & 100{\cdot}00 \end{aligned}$$

The calculated nitrogen is higher than the quantity found by analysis. Should the gelatin be found really to contain some sulphur then this element would have to be substituted for some oxygen in the formula. Gerhardt, who is perhaps the only author who has noticed this speculation of Hunt's (Traité de Chimie, 4, 508), put it to a test and made an interesting discovery. He caused isinglass to boil during some days with dilute sulphuric acid, and obtained ammonic sulphate and a considerable quantity of sugar (gelatose or ichthyocollose) which by fermentation was transformed into alcohol and carbonic acid. (Comp. Gerhardt, Chim. Organ. Appliq. à la Physiologie Vegetale de M. Liebig, p. 287.)

In the case of gelatin, as in those of the albuminous substances, the products of chemolysis which are crystalloids have been well studied, but those which are colloids, at least in the first instance, have been neglected. Scheele already, in attacking gelatin with nitric acid and heat, obtained oxalic and malic (saccharic) acid (questioned by Gerhardt, but without his giving grounds), a greasy and an astringent matter. (Berzelius, Lehrb. der Chem., 3rd edit., 9, 800.)

In consequence of some late researches on glutin, it has been proposed to distinguish and separate isinglass from ossein proper, and therefore

it is not safe to conclude from Gerhardt's experience that glutin from bones and other tissues of higher animals will also yield a fermentable sugar.

Gelatin (like pure cellulin) when purified by dialysis is imputrescible in the ordinary sense, perhaps because being free from salts it affords no food to the organised putrescence ferments. It combines with colloid acids, and therefore Graham termed it a colloid base.

Keratin, or horn-substance, the constituent of all kinds of coverings of the higher animals, exists in epidermis, epithelium, in hair, horns, nails, claws, hoofs, and feathers, mixed with a very great variety of colouring ingredients, as great perhaps as that of the colouring ingredients of the chitin of articulates. The crystalloids obtained by its chemolysis which are the best known are leucin, tyrosin, aspartic acid. The *aperçus* of similarity to amylonides in the bearing of keratin are numerous, but not yet sufficiently precise to be used. But most remarkable is the bearing of the serpent's skin. This organ when treated alternately with cold oil of vitriol and cold potash ley, whereby nitrogenous substances are dissolved, leaves a substance which is isomeric with cellulin, and yields fermentable sugar on boiling with dilute acids. (De Luca, Compt. Rend., 57, 437.) Now this cellulin might be there as such, and not as a product of decomposition of keratin as is supposed by treating it in this place. It would then be analogous to the occurrence of cellulin in the skin of the silkworm, and the brown remains of the pupas which are left in the cocoons when the butterflies escape, and are capable of yielding a substance isomeric with cellulin which may be converted into glucose. When the caterpillars are boiled for several hours with strong hydrochloric acid, and this treatment is repeated three times with the residue, and the residue from the processes is washed with strong potash ley, then with water, and dried between 100° and 110°, a white light substance nearly free from nitrogen is obtained which gradually diffuses in oil of vitriol, forming a gummy, colourless liquid. This solution added by small quantities to boiling water, and boiled for an hour or two, yields fermentable sugar, which further reacts like glucose with common salt and with potassio-cupric tartrate.

These forms of cellulin then might be admixtures of the keratin and chitin tissues respectively, and then they would show nothing as regards the chemical structure of the keratin. But even so the occurrence of cellulin in the skin of the serpent is a powerful argument in aid of the hypothesis of the amylonide structure of organic tissues. But it is of little consequence if we possess at present no direct evidence of the presence of an amylonide radicle in keratin, and in gluten from ossein and other substances. For as they are derived from the higher albumin, and as albumin does indubitably contain an amylonide radicle, it is fair to assume that the derivates also may contain it, unless indeed they have lost the amylonide radicle in the course of their being changed into structure elements. But this is not likely, as their composition and bearing is too much like albumin.

We now come to the class of albuminous substances in which the presence of an amylonide radicle can be proved physiologically by biolysis much better than by chemolysis. Of these albuminous bodies we have the white of egg, or albumin; the albumin of blood serum; the albumin of flesh; the contractile matter of flesh, or syntonine; and the curdling matter, or myosin; then paraglobulin and fibrin as natural educts from blood or other serous liquids; then casein from milk, with which some declare paraglobulin to be identical; then globulin from the crystalline lens, and globulin from hematoglobulin,

or hematocrystallin, and some abnormal products of disease not yet sufficiently studied.

All these by chemolysis yield products of which the greater number are crystalloids and well understood, while another part is not yet fully interpreted.

Bopp had found that when any of the albuminous substances is boiled with strong mineral acid it yields a mixture of products containing tyrosin, leucin, leucimide, and some other crystalloids in small quantity; the noncrystallisable portion tastes sweet, does not undergo fermentation with yeast, evolves hydrothion when boiled with potash and afterwards acidified with acetic acid, and forms a violet solution with potash and cupric sulphate, or potassio-cupric tartrate.

These inquiries were subsequently extended, and compound volatile alkalies, fixed alkaloids, such as fluorescentin and fluopittin, a substance recalling the bearing of biliary matters by its spectrum, and others in smaller quantity were discovered.

In these chemolyses the process did probably go beyond the stage at which the amylonides are, or one of them is, separated but not destroyed. For Schützenberger (Chem. Centrbl., 1875, p. 614 and seq.) decomposed albumin by boiling it for one hour only with very dilute sulphuric acid, and found amongst the soluble products a nitrogenous body which strongly reduced Fehling's solution, was precipitated by ammoniacal lead acetate, gave no precipitate with mercuric nitrate, and seemed to him to be glucose or some such body. He further decomposed albumin with barytic hydrate (l.c., p. 631) under pressure at a temperature of from 150° to 200°, and obtained by various manipulations, a considerable quantity of matter, 3 to 4 grm. from 70 grm. dry coagulated albumen, which had a clearly sugar-like taste, did not evolve ammonia when heated with potash, was soluble in water, but insoluble in absolute alcohol. When heated on platinum foil it swelled up and charred without giving any sublimate, and smelt like charred bread. This product, fully exhausted with alcohol, contained—

		Found.	Calculated from theory given below.
		Per-cent.	Per-cent.
(*a.*)	C	44·2	44·7
	H	7·6	7·7
	N	14·8	15·0

Another similar product obtained in the same manner from hemi-albumin, contained—

(*b.*)	C	42·4
	H	8·0
	N	14·5

From this statement it appears that one such reducing body was obtained from albumin by the side of hemi-albumin under the influence of sulphuric acid, another from hemi-albumin itself by secondary chemolysis with baryta. Two such nitrogenous copper reducing nuclei seem therefore to be present in albumin.

Schützenberger considers the body (*a*) to be a compound of $C_{12}H_{28}N_8O_4$ with $3C_4H_8NO_2$ (glykoprotein *a*) — H_2O. The compound $C_{12}H_{28}N_8O_4$ or $C_6H_{14}N_4O_2$ he explains as an amide of cellulin; and he thinks that from this body might be derived the small amount of dextrin which was found in a third experiment on the chemolysis of hemi-protein by baryta. In this experiment a mercuric

nitrate precipate yielded on decomposition matters from which absolute alcohol extracted a sugar-like amorphous body, which contained—

	Exp. III. *a.*				Exp. III. *b.*	
	1.	2.	3.	4.	5.	6.
C	49·51	48·79	—	—	52·8	—
H	6.78	6·86	—	—	6·83	—
N	—	—	13·45	12·8	—	14·24

Beside this nitrogenised body the baryta salt (in experiments like Exp. III.) always contains a certain quantity of a non-nitrogenised body, which is insoluble in alcohol, is precipitated by ammoniacal lead acetate, and by boiling with dilute sulphuric acid is metamorphosed into a body which reduces Fehling's fluid. The original body does not reduce Fehling's fluid, but gives oxalic acid on treatment with nitric acid. The body gave on analysis—

C	42·8
H	6·56

These figures are near those which would be yielded by dextrin. Schützenberger surmised that the occurrence of this cellulin-like body in the baryta chemolysis stood in connexion with the occurrence of a body reducing Fehling's fluid which was formed in the sulphuric acid chemolysis. But although he thus recognised that "the molecle f albumin contains a small quantity of a cellulin amide," he neglected this ingredient in all his following calculations and theories, and came to the result that albumin was a complex ureide; the further details of structure he explained upon this basis, and in consequence did not arrive at the explanation of the structure of albumin as we shall have occasion to formulate it.

The amylonide nature of the albuminous substances is proved by the so-called glycogenetic function of the liver, and the lactopoetic function of the mammary glands of the carnivora. It is further proved by the growth and development from eggs to the adult state of maggots reared on boiled white of egg exclusively. These remarkable proofs science owes mainly to Bernard; but he did not draw the conclusion which is here propounded, and like other inquirers in his time, considered the formation of sugar from albumin an accidental vicarious act performed when no carbo-hydrates were at hand, to suit the wants of the body.

The paralbumin of Scheerer obtained from ovarian cysts contains, besides albumin, a body resembling liver-starch in this, that it dissolves in water to a turbid fluid, is precipitated by alcohol, coloured brown by alkalies and yellow by iodine, and when boiled with dilute sulphuric acid yields a product which reduces cupric and bismuthic oxide. At the same time there is an albuminous body present which can be precipitated by neutralisation and boiling; and the liquid contains another probably albuminous body, which on boiling with mineral acids yields a flocculent brown precipitate and a solution reducing after neutralisation copper, bismuth, and indigo. The original substance gave by analysis 49·77 % C, 7·6 % H, and 7·4 to 8·8 % N. This body is therefore an amylonide, similar to the lower forms of mucin; but as a pathological product it deserves special study.

The following list of organo-plastic substances is so arranged that it begins with the simplest, namely, cellulin, and ends with the most complicated, *i.e.*, albumin. As they are considered here principally in their hypothetical character of substances formed upon the type of amylon,

they are termed amylonides, and the sugars to which they give rise or are supposed to give rise by chemical or physiological agencies are named in connexion with them on the right of each term.

Organoplastic Animal Amylonides, and Sugars to which they give rise.

Cellulin	Dextrose.
Glycogen, or hepatic amylon	Dextrose.
Tunicin	Dextrose.
Chitin	Chitose.
Conchiolin	Conchiolose.
Hyalin	Hyalose.
Fibroin	Fibrose.
Serin	Serinose.
Spongin	Spongose.
Keratin	Keratose.
Chondrin	Chondrose.
Cerebrin	Cerebrose.
Phrenosin	Cerebrose.
Kerasin	Cerebrose.
Gelatin	Gelatose.
Ichthyocollin	Ichthyocollose.
Mucin	Mucinose.
Paralbumin	Paralbuminose.
Albumin	Albuminose, or albumino-dextrose, or lactin.

These names seem convenient as designating the origin of each variety of sugar; they or any of them will have to be changed as soon as it can be shown that the sugar corresponds to a known variety of more general occurrence. Some terms may be doubtful, *e.g.*, the reducing body from serin or silk gelatin, may have been sericin, which is not a true saccharoid, but a hemi-saccharoid or glycerenide, and thus resembles the fats and phosphorised substances of the brain and other tissues. Again, in the case of the sugar obtained from gelatin, it might be possible that one of the reducing amido-acids (glutamic and asparaginic) had simulated sugar, a doubt which requires further attention. This doubt could, however, not apply to the sugar obtained from isinglass, which did not only reduce but by fermentation gave alcohol.

Hepatic amylon or glycogen occurs twice in the list, namely, as an organoplastic substance by itself, and giving rise to dextrose; and as a product of the decomposition of albumin, as albuminose or albuminous dextrose. Even if these bodies should be identical, which is not very likely, the repetition will be a concession to caution, as they might be formed in different places by different processes (lactin in the mammary gland, glycogen in the liver). Some of the sugars differ greatly from each other at first sight. Supposing the properties of dextrose to be well known, we can compare the other bodies to it. The sugar from chondrin (chondrose) turns the plane of polarised light to the left. Lactin turns to the right, but is a disaccharoid, one molecle of it yielding by hydration two molecles of lactose. Cerebrose is isomeric with dextrose, reduces copper, turns the ray of polarised light to the right, and crystallises from water in anhydrous crystals, while dextrose crystals contain a molecle of water.

The chemical constitution of the fats and of the phosphorised substances is determined by a radicle which by chemolysis to ultimate

crystalloids appears as glycerin. The radicle receives additional importance in relation to our present argument by the fact that it can be considered as a half-sugar so to say, a hemi-saccharon. Its formula is about half that of sugar; its function is that of a tridynamic alcohol, while the most developed sugar is hexadynamic alcohol. Moreover, two molecles of glycerin (so it has been stated) may combine and form sugar. The substitution of three molecles of fatty acid for three molecles of hydroxyl in glycerin produces the vegetable and animal fats. In the phosphorised substances, however, only two molecles of hydroxyl are substituted by fatty acids; the third hydroxyl is substituted by phosphoric acid, and to this is attached a collateral chain of smallest radicles, the complex of which is called neurin or cholin. Fats and phosphorised substances may therefore be classified as hemi-saccharonides or glyceronides or as ethers of the alcohol glycerin with simple or compound acids. To this class of bodies serin or silk gelatin may possibly belong; it contains and yields by chemolysis a glyceritic amide (sericin) serosamide, which is analogous to chitosamide thus far that we might signalise it by the term hemi-chitosamide were not the amidogen deficient in the ideal second half of the ideally severed saccharoid radicle.

No. II.—Chitin, the simplest Nitrogenised Organoplastic Amyloniде.

Introduction.—Chitin forms the principal constituent of the dermatic envelope and entire firm skeleton, tendon-like parts, and attachments of muscles of nearly all articulate animals, and of insects not only in the fully developed, but also in the larval and pupal state. It is also said to be present in the lining membranes of the tracheæ of air-breathing articulates, and forms part of the gills of those breathing water; it is also a constituent of the lining membrane of the intestinal canal. It possesses great flexibility when it forms the external organs, membranes, and hairs of animals by itself, combined with only little inorganic salt and organic colouring matter; but it acquires great hardness by an impregnation, during the course of its structural development, with carbonate and phosphate of lime, and then as dermatic skeleton shows analogy with the ossein of the skeleton of vertebrate animals.

As the external envelope of animals of the class of articulates is formed gradually and is changed repeatedly during their growth and life, it is advisable to bear in mind that the chitin obtainable by chemical proceedings may not be the same in the same species at all times, and that it may also differ in different species. The bones of young vertebrates yield chondrin, those of adults ossein; the growing antlers of the stag yield chondrin, only when just full-grown they yield chondrin and ossein, and after maturation and scouring ossein only.

Modes of Preparation.—The materials which yield chitin with the least trouble are perhaps, in places near the seashore, the transparent crustaceans such as shrimps, prawns, and langostinos of Cadiz, but in inland countries the wing-cases of coleoptera. If entire insects are taken it is necessary to remove mechanically the internal organs, particularly the intestinal canal, to exclude the possible admixture with the chitin of any cellulose from leaves or other vegetable structures which have served as food. The shells of larger aquatic crustaceans are also a convenient and easily procurable material, although they require prolonged treatment with much acid for the removal of the calcic carbonate and phosphate.

The materials selected are exhausted by boiling with caustic soda solution, prolonged treatment with dilute hydrochloric acid, and second

boiling with caustic soda, then with water and boiling alcohol in succession. If the chalky shells of large articulates are used, the following succession of operations may be adopted. The shells are pounded in a metal mortar until all tubes are crushed and all stiff parts comminuted. The pasty mass is now boiled with strong caustic alkaline ley, and the latter is repeatedly changed; when all matter soluble in alkali is removed, the well-washed mass is placed in dilute hydrochloric acid and treated with this agent until no further effervescence is perceived on standing, and until all particles are soft and translucent. The residue is now washed and boiled with spirit until all colouring and other soluble matter is extracted. The chitin thus obtained is dried on the water bath and shrinks to a tough, shrivelled, greyish-white mass of particles like parchment shreds. These may be ground in a coffee mill. Chitin thus prepared may be purified, according to Städeler, by boiling it for 12 hours or longer with a mixture of one volume of oil of vitriol and four volumes of water, when a part of it is changed into soluble crustacin (alkaloid) and chitose (sugar), while another part remains as a swollen pasty mass, being almost pure chitin free from ash. This may be washed by decantation as long as it contains free acid, but as soon as all acid is removed it remains partly suspended in the liquid; the latter must therefore be evaporated to dryness.

When Peligot treated the skins of silkworms with caustic ley alcohol, ether, and acetic acid, he obtained translucent bags open at both ends, consisting of a chitin which contained 48·13 % C, 6·90 % H, 8.30 % N, and 36·67 % O.

After this had been digested with dilute permanganate of potash it contained 47·38 % C, 7·02 % H, 6·15 % N, and 39·45 % O.

When this was treated with sulphuric acid containing 6 molecles of water, 44 % of its weight was transformed into soluble products, while the residue still contained 5·8 % nitrogen. The latter was then treated for several hours with hot, or for several days with cold, concentrated watery solution of potassic permanganate, then with potassic bisulphite, and finally washed with water. The remaining white mass did not evolve ammonia when boiled with potash ley; but when it was made into a paste with potassic hydrate and water, and heated to 100° C. for eight days, it dissolved, evolving ammonia on the one hand and forming fatty acids on the other, which were set free by acids. Even after two-thirds of the chitin had thus been decomposed, the remainder recovered from the alkali still contained 6·2 % of nitrogen, and continued to evolve ammonia on renewed application of potash. When this chitin, after having been softened in water, was treated with oil of vitriol and iodine, it exhibited under the microscope brown spots and blue particles of irregular shape. The blue colour became more plainly visible if the skin was left for some days in the iodine solution and then moistened with oil of vitriol, whereupon entire pieces of the membrane became greenish at first, but changed to indigo blue as the iodine evaporated. From this chitin, moreover, watery cuprammonia extracted cellulin which was precipitated from the solution by acids. The horny substance of the lobster also exhibited the reaction of cellulin with oil of vitriol and iodine. Städeler declared this chitin to have been impure, as he himself could extract nothing from pure chitin by cuprammonia. But it must be borne in mind that this impurity could only have arisen from a structural admixture of cellulin with chitin, and therefore chitin which gives this reaction is unsuitable for experiments on pure chitin, as at present chitin and cellulin cannot be separated by the means ordinarily adopted for the purification of chitin.

Properties.—Chitin, when purified by boiling with dilute sulphuric acid or by solution in concentrated hydrochloric acid and precipitation with water, is a white amorphous mass sometimes tough and difficult to reduce to powder.

Composition.—Its elementary composition has been estimated by many observers with the following results:—

		C.	H.	N.	O.
Children and Daniell.	Todds, Cyclop. of Anat. and Physiol., 2, 282.	46·08	5·96	10·29	37·67
K. Schmidt	(Mean of 15 combustions)	46·66	6·60	6·53	40·21
„	maxima	46·80	6·77	6·79	—
„	minima	46·48	6·43	6·32	—
Städeler		46·32	6·40	6·14	41·14
Lehmann, 1, 382	(Mean) Lehrb. 1853,	46·734	6·594	6·493	40·179
Ledderhose	(Mean of 12 combustions)	45·69	6·42	7·00	—
„	mean of analyses with more carbon	46·026	6·256	7·00	—

K. Schmidt had calculated the empirical formula $C_{17}H_{28}N_2O_{11}$ from his analyses having deducted from 0·6 to 2·0 °/₀ of ash (chitin from crustacea). Schmidt's formula was $C_{17}H_{14}NO_{11}$ when C = 6, O = 8; on account of its decomposition by heat and acids, he was of opinion that chitin contained the elements of the primitive fibres of the muscles of arthropodes $C_8H_6NO_3$ with a carbohydrate $C_9H_8O_8$. But Städeler calculated the formula $C_9H_{15}NO_6$, which being more simple and apparently supported by the decomposition of chitin into a glucose and a hypothetical amido, acid was more generally adopted. Its theory agreed with Städeler's results as follows:—

	Theory.		Städeler.
9 C	108	46·35	46·32
15 H	15	6·44	6·40
N	14	6·01	6·14
9 O	96	41·21	41·14

The nitrogen had been found much higher by Children and Daniell, namely, 10·29 °/₀; Payen found 8·99 °/₀; Schlossberger, 6·4 °/₀ N. Bütschli and Emmerling (Du Bois and Reichert's Archiv. 1874, 362) estimated the nitrogen in chitin as the mean of three experiments 6·32 °/₀. Emmerling (loc. cit., p. 370) analysed chitin, which had been precipitated from hydrochloric acid solution by water, and obtained by combustion with soda-lime, 6·24 °/₀ by combustion with cupric oxide 7·02 °/₀ N. In the last analysis a loss is noted to have been incurred. Ledderhose estimated the nitrogen in chitin twice by combustion with soda-lime, observing the precautions otherwise known but lately again pointed out by Makris (Ann. Chem. 184, 371), and obtained 6·96 and 7·049 °/₀ or mean 7·00 °/₀ nitrogen.

With the aid of these later data concerning the per-centage of nitrogen, Ledderhose came to the formula $C_8H_{13}NO_5$, which requires—

C	47·29
H	6·40
N	6·896

but thinking that the amount of carbon required by it was too high as compared to that found by analysis, he preferred $C_{15}H_{26}N_2O_{10}$, which requires—

		Mean of all his analyses.
C	45·685	45·69
H	6·598	6·42
N	7·10	7·00

In the endeavour to support this formula by an equation intended to express the structure of the body he unfortunately fell into an error, whereby $C_{15}H_{26}$ in the left half of the equation are supposed to be counterbalanced in the right half by $C_{18}H_{38}$. The error is not a misprint, but is stated in words printed in emphatic type (p. 224–225), and the supposed equation is repeated twice over. That chitin was free from sulphur and phosphorus, was expressly proved by Schmidt.

Decompositions.—When heated in the open air chitin does not change in form, but simply chars without evolving carbonate of ammonia. But when subjected to thermolysis in a closed apparatus it gives out water, acetic acid, ammonic acetate, and a small quantity of an empyreumatic oil, while a charcoal of the original form of the chitin remains. Under the influence of air and water it is only slightly altered even after long periods; thus Schlossberger kept some chitin under water for a year, and found it only partly dissolved, the other part changed into a slimy mass. Long boiling with water does not alter the purified skins of silkworms, but the water takes up a small quantity of a nitrogenous substance, which, when the solution is evaporated to a small bulk, is precipitable by tannic acid, chlorine, or alcohol. Owing to this reaction Lassaigne believed that some gelatin had been formed from the skins; but this has probably no relation to the chitin itself, but was only the effect of the adhesion to the skins of, or penetration of its structure by, a collogenetic matter not yet isolated. When Schmidt heated chitin with water in a sealed tube to 280°, he observed it to become brown and brittle, but not to alter otherwise. Chitin dissolves readily in oil of vitriol, forming a thick solution; this gives a copious precipitate when thrown into water immediately or within 12 hours. The solution in oil of vitriol is colourless at first; on standing it deposits a few dark flakes. I have not observed it to become black after 12 hours, but Schmidt saw it become black after 48 hours, and perceived that it had deposited a small quantity of insoluble matter, had a choking smell, and contained acetic acid and an ammonium salt. The colourless solution of chitin in oil of vitriol may after standing some hours be diluted, and then yields a precipitate of unchanged chitin; but if it has been heated and decomposed, it yields, after neutralisation with baryum carbonate, a soluble baryum salt (acetate), and reduces alkaline copper solution (chitose). Berthelot dissolved chitin in oil of vitriol, and dropped the solution into a hundred times its weight of water. He then boiled for an hour and neutralised with chalk; on evaporation he obtained a sugar which could be fermented into alcohol and reduced alkaline copper solution. Städeler now gave to this experience a more precise explanation. He boiled chitin with a mixture of one volume of oil of vitriol and four volumes of water for 12 hours or longer, separated what remained insoluble, supersaturated with lime (whereupon ammonia was evolved) and evaporated the filtrate to a syrup. This remained amorphous, and reduced cupro-potassic tartrate. No tyrosin, leucin, or glykokoll could be obtained from the syrup, but it contained a small quantity of amorphous matter, the nature of which he failed to ascertain. He

formulated the splitting up of chitin as follows, overlooking entirely the formation of acetic acid first found by Schmidt :—

$$C_9H_{15}NO_6 + 2H_2O = C_6H_{12}O_6 + C_3H_7NO_3.$$

(Sericin is $C_3H_7NO_3$.)

Chitin is soluble in cold concentrated hydrochloric acid without change of colour. The skins of insects when immersed in hydrochloric acid containing six molecles of water become transparent, are disintegrated, and after a few minutes dissolved. After neutralisation of the solution by alkalies, tannic acid throws down a precipitate containing nitrogen (crustacin ?). The shells of crustaceans are never entirely soluble in hydrochloric acid in the cold.

The solution of chitin in concentrated hydrochloric acid is not changed quickly in the cold, but deposits the entire amount of the chitin which it contains by suitable dilution with water. When the hydrochloric solution is mixed with alcohol, two bodies free from nitrogen are obtained, the nature of which has not been ascertained by their discoverer, Bütschli. One part of purified lobster-shell dissolves in 12 parts of hydrochloric acid at 40° C., and is immediately chemolysed. A cold hydrochloric solution, on being boiled for some time, becomes blackish-brown, and after an hour's boiling the chitin is completely chemolysed. If it is now evaporated on the water-bath a compound crystallises in large quantities, which has been described by Ledderhose as hydrochlorate of glucosamin, and to which he ascribes the formula $COH\ (C\ H\ OH)_4\ CH_2\ NH_2 + HCl$, or $C_6H_{13}\ N\ O_5 + HCl$, being that of a carbohydrate in which one group OH is substituted by NH_2, while a molecle of HCl is in so-called molecular combination. The crystals are mixed with a black humus-like matter which is completely insoluble in water, and can be separated from the crystals after expulsion of all free hydrochloric acid by repeated crystallisation of the salt from water. The quantity of the new salt containing the new base (which, as an amide of a previously known body chitose, we will term chitosamin), obtainable from a given weight of chitin, was estimated as follows :—A weighed portion of chitin, dried at 110°, was boiled during an hour with concentrated hydrochloric acid and evaporated to dryness. The residue was dissolved in water digested for some time on the water-bath, and freed from black flakes by filtration. The solution was again evaporated, dried at 100°, and weighed. The product amounted to 91 % of the chitin employed. The salt lost but little in weight by a repetition of the purifying process. The amount of chitosamin formed from chitin may therefore be calculated, deducting the hydrochloric acid and allowing for some impurity, to be from 70 to 75 % of the chitin employed. The solution contains no ammonic chloride, so that it seems as if the entire amount of the nitrogen contained in chitin was transferred into the chitosamin. No oxalic acid is formed, and it seems as if chitosamin was the only fixed product of the chemolysis. The solution of chitin in concentrated hydrochloric acid is subjected to distillation to collect the volatile products. The distillation is repeated after addition of water to the liquid in the retort. The distillates are neutralised with silver oxide, the dissolved silver is removed by hydrothion, and the acid filtrate is neutralised with barytic carbonate and evaporated. A crystalline salt is obtained, having the character of acetate. But there is a small amount of a higher fatty acid present, probably butyro-acetic, which depresses the baryta a little and can be recognised by its peculiar smell. By a comparison of the results of Berthelot with his own, Ledderhose comes to the result that chitin by treatment with concentrated sulphuric acid is chemolysed in the same manner as by concentrated hydrochloric acid, giving chitosamin and acetic acid ; but when these products are boiled long in very dilute solution, the chitosamin is further split up into fermentable sugar and ammonia.

The decomposition of chitin under the influence of acids and heat might therefore find its simplest expression in the formula—

$$C_8H_{13}NO_5 + 2H_2O = C_6H_{13}NO_5 + C_2H_4O_2.$$

The small amount of butyric acid obtained seems to indicate that with the simplest chitin, aceto-chitin, there occurs mixed a butyro-chitin, which contains the radicle butyryl in the place where the simplest chitin contains the radicle acetyl.

Chemolysis of Chitin with Sulphuric Acid.—20 grms. dry pure chitin were heated in a sealed leaden tube with 360 c.c. of sulphuric acid of 10 % strength for 48 hours. The liquid was then found clear and to contain a reducing body and acetic acid, but a large proportion of the chitin was only softened, of the consistence of cheese, another flaky. The liquid was filtered off and the solid residue treated with oil of vitriol. It dissolved, forming a transparent thick fluid. This with water gave a copious white precipitate with water. All the portions of liquid and precipitate were boiled for eight hours in a platinum still, and three litres of distillate were drawn off, one after the other, water being added after each litre drawn off. The liquid in the still contained yet much undissolved and apparently undecomposed chitin. The sulphuric acid solution gave a precipitate with phosphotungstic acid. The undecomposed chitin was treated with fresh oil of vitriol, and the solution treated with water. Pure white chitin was precipitated which, after washing with water and absolute alcohol, appeared as a white amorphous mass like starch. The solution contained an alkaloid *crustacin*, precipitable by phosphomolybdic acid and a sugar, chitose.

Chitin is coloured orange-yellow to dark brown-red by iodine water; addition of oil of vitriol to this test causes the chitin to dissolve, but does not produce either a violet or a blue coloration. Concentrated nitric acid dissolves chitin without discoloration; one part of chitin dissolves in an equal weight of cold nitric acid containing four molecles of water. By the addition of a large amount of water chitin is again precipitated, but some acetic acid is always formed, the more the longer the action of the acid has been continued. Payen observed that crabs claws immersed in nitric acid became transparent after a few moments; when the acid was drained off the membrane retained at first its original form but afterwards deliquesced to a colourless liquid, beginning at the edges. Alkalies in the dilute or even pretty concentrated state alter or affect chitin so little that they can be employed for its purification. The alkali may be as strong as one part of potash hydrate in three parts of water, and the chitin may be boiled in this for days, or even heated with it to 210° in sealed tubes without being sensibly attacked; but when it is fused with solid hydrate of potash it evolves ammonia (Payen) and hydrogen (Ledderhose) without charring, and leaves a residue soluble in water. If the fusion is not continued until the mass is white, but interrupted, and the fused mass is dissolved in water mixed with sulphuric acid and distilled, acetic and a little butyric acid are obtained and recognised by their baryum salt.

Frémy's Chitin, free from nitrogen (Compt. Rend., 39, 1052, N. Ann. Chim. Phys., 48, 1855, 93). The skeletons of crustacea are treated with cold dilute hydrochloric acid, the undissolved residue is boiled for several hours with potash ley, and then washed with water, alcohol, and ether. A transparent horny substance is thus obtained, containing on the average 43·35 % C, 6·65 % H, and 50·00 % O, but no nitrogen, and therefore isomeric with cellulose ($C_6H_{10}O_5$). This substance resists the influence of dilute acids and alkalies; acids in particular do not convert it into sugar, and fuming nitric acid has no action on it whatever. By other concentrated mineral acids it is broken up, dissolved, and converted into an acid which Frémy compares with his metapectic acid; by boiling nitric acid he obtained oxalic acid.

Rouget's Chitin and modified Chitin.—(Compt. Rend., 48, 793). The dermatic envelope of the crustacea in their natural state, or dissolved in hydrochloric acid when treated with mercuric nitrate, gives a rose-red coloration; when treated with nitric acid and ammonia an orange-red coloration; and when mixed with iodo-chloride of zinc a violet reaction. (Zinc chloride solution, sp. gr. 1·8 at 15° 100 parts; potassic iodide 6 parts to be dissolved in the syrup, and then as much iodine as the mixture will take up.) The skins after ebullition with potash ley of 40°, which does not alter their appearance or structure, react more quickly with the iodo-chloride of zinc, producing a more intense bluish-violet colour, but they remain still unchanged by caustic-potash ley, acetic or tartaric acids. Boiled for half an hour with five times their weight of potassic hydrate and such a quantity of water that the mixture would immediately solidify if allowed to cool, a large quantity of ammonia is liberated and half the chitin is dissolved. The residue of modified chitin shows traces of the microscopic structure of the original tissue, is translucent, gelatinous, and easily broken up while moist; in the dry state it is greyish white, dull in appearance, and light in weight. With tincture of iodine or with iodine and dilute acetic acid, this residue becomes violet throughout; iodo-chloride of zinc colours it pure blue. It dissolves almost instantly in acetic or tartaric acid, or in warm water containing 0·5 % of hydrochloric or nitric acid, and is precipitated from these solutions by alcohol or by caustic ley as a somewhat translucent paste which dries up to a yellowish translucent gum. The acid solution and the precipitate are coloured reddish-violet by a few drops of solution of iodine in potassic iodide. It dissolves in oil of vitriol with yellow or brown colour, and is precipitated by water from the freshly-prepared solution as a white powder. The solution is less completely precipitated by water after having been allowed to stand for 12 to 24 hours, and is then found to contain sugar. After being precipitated by alcohol or alkalies from its solution in acid it is still soluble in acids and contains nitrogen.

Purified chitin is insoluble in water, aqueous ammonia, acetic acid, alcohol, and cuprammonia. The dark amorphous humus-like matter obtained in the chemolysis of chitin remains to be investigated.

Chitosamin, $C_6H_{13}NO_5$, is obtained from the sulphate by treatment with barytic hydrate without employment of heat. It crystallises from alcohol in needles. The nitrate and sulphate are obtained by treating the hydrochlorate with nitrate or sulphate of silver; both salts crystallise in fine needles.

A double salt of chitosamin and lead chloride crystallises from water.

The hydrochlorate is obtained directly from chitin in the manner above described. It turns the plane of polarised light to the right. The limited or so-called specific rotation, according to Ledderhose, is + 70°; according to observations made by me, it is + 72° 15′. The rotation is more considerable immediately after solution, and falls gradually during 24 hours, after which it becomes stable at the points related.

One hundred parts of a saturated solution of chitosamin hydrochlorate contain 28·83 parts of salt dry at 100° C.

All compounds reduce potassio-cupric tartrate, but do not ferment with yeast.

Behaviour of Chitin in various Species of Animals.—As to organs, see below; also K. Schmidt, Zur vergl. Physiol. der wirbellosen Thiere, Braunschweig, 1845; Frey and Leuckart, Wagners Zootomie, 2, 133, and 167; Grube, Wiegm. Archiv. 1853, 104; Leydig, Müller's Archiv., 1855, 376. On other (problematical) statements concerning the occurrence of tissues containing chitin in other nonverte-

brates compare Schlossberger Vers. etc. Thierchemie 1856, 1, 1, 230. Leyer and Köller (Ann. Chem. 83, 336) and Schlossberger (loc. cit. 228) when chemolysing entire wing-cases of cockchafers obtained leucin and tyrosin. This therefore was due to a constituent other than chitin. The crusts are therefore a mixed tissue. Schmidt examined chitin from Melolontha vulgaris, Ateuchus sacer, Astacus fluviatilis, Astacus marinus, Squilla mantis and found the elementary composition as stated above. Schlossberger (loc. cit., 227) examined the shell of Palinurus and found in its chitin 6·4 % N.

The shells of crustacea contain up to 63 per cent. of organic matter; the mineral ingredients are mainly carbonate and phosphate of calcium.

Thorax of crayfish contains organic matter 33·3 %; calcic carbonate with little sodic chloride, iron, manganese and pigment 61·0 %; calcic phosphate 5·7 %. (John, Chem. Schriften, 3, 49.)

Crayfish shells: organic matter 28·0 %; calcic carbonate 60·0 %; calcic phosphate 12·0 %. (Merat and Guillot, Ann. Chim. 34, 71.)

Crayfish thorax: chitin 46·73 %; ash 53·27 %. In this ash 86·83 % calcic carbonate and 13·17 calcic phosphate. (K. Schmidt, l.c.)

Shell of crayfish: Organic matter 36·5 %; calcic carbonate 56·8 %; calcic phosphate 6·7 %. (Frémy, l.c.)

Shell of crab: Organic matter and water 28·6 %; sodium salts 1·6 %; calcic carbonate 62·8 %; calcic phosphate 6·0 %; magnesic phosphate 1·0 %. (Goebel, Schweigger's Jour., 39, 440.) The same inquirer found in the claws of crab: organic matter 17·18 %; calcic carbonate 68·36 %; calcic phosphate 14·06 %. In the "teeth" of the claws: organic matter 12·75 %; calcic carbonate 68·25 %; calcic phosphate 18·75 %. Shell of crab: organic matter 28·0 %; calcic carbonate 62·80 %; calcic phosphate 6·0 %. (Chevreul, Ann. gén. des Sci. Phys., 4, 124. Schweigger's Journ. 32, 495.)

Shells of squilla mantis: chitin 62·84 %; salts 13·17 (of these calcic phosphate formed 47·52, and carbonate 52·48.) (K. Schmidt, loc. cit).

Claws of lobster: Organic matter and water 44·76 %; sodium salts 1·5 %; calcic carbonate 49·26 % calcic phosphate 3·22 %; magnesic phosphate 1·26 %. (Chevreul, l.c.)

Claws of lobster: Organic matter 28·0 %; calcic carbonate 40 %; calcic phosphate 14·0 %. (Guillot, l.c.)

Shell of lobster: Organic matter 44·3 %; calcic carbonate 49·0 %; calcic phosphate 6·7 %. (Frémy, l.c.)

Summary of Historical Literature.—Chitin was first distinguished from horn-substance by Odier in 1823, and was supposed by him to be free from nitrogen (Mem. Soc. d'Hist. Nat. de Paris, 1, 29). Lassaigne recognised that it, named by him entomoderm, contained nitrogen (Compt. Rend., 16, 1087; Journ. Chim. Méd., 19, 379). It was next studied by Payen (Compt. Rend., 17, 227), and more intimately by K. Schmidt (Ann. Chem., 54, 298). Schlossberger (Ann. Chem., 98, 105) contributed to its more accurate knowledge by defining the organic matter contained in some shells and in the byssus of shells which had been confounded with chitin, as conchiolin. Berthelot (Compt. Rend., 247, 727, and in detail N. Ann. Chim. Phys., 56, 149) found that chitin on chemolysis with acid gave a kind of sugar. Peligot (Compt. Rend., 47, 1034; N. Ann. Chim. Phys., 58, 83) in consequence endeavoured to find the amylum-like body in chitin which gave the sugar. Städeler (Ann. Pharm., III. 21) also studied chitin, and gave a new formula differing from that first given by Schmidt. G. Ledderhose (Zeitschr. Physiol. Chem., 2, (1878) 213) found the sugar-like body to contain nitrogen, and termed it glykosamine. He also proposed a new formula for chitin, which has been discussed in the foregoing.

No. 4.

Report on Pyæmia, Septicæmia, and Purulent Infection.

Report to the **Pathological Society** of **London** by the **Committee** appointed "to investigate the **Nature, Causes**, and **Prevention** of those **Infective Diseases** known as **Pyæmia, Septicæmia**, and **Purulent Infection**."

We beg to submit the results of the investigation into the nature and causes of those infective diseases known as pyæmia, septicæmia, and purulent infection undertaken at the request of this Society in accordance with an engagement entered into by the Society with the Local Government Board.

The plan of investigation followed by your Committee has been:

1. To collect statistical information with respect to the affections in question from nearly all the large London hospitals during the decennial period from 1869 to 1879, with a view of determining the history of the disease in the surgical wards of the metropolitan hospitals during that decennium.
2. The collation of 155 detailed reports of cases, with the object of attempting to classify them according to their clinical and pathological features, and of ascertaining how far these correspond with the diseases produced experimentally in animals.
3. An examination of a considerable number of microscopical preparations of the various organs of the body from typical cases, together with observations on the blood and urine, in some cases made during life, to illustrate the nature of the pathological changes in different stages of the various septic diseases.

PART I.

(1.) *Sex and Age.*—Out of 321 cases of pyæmia and septicæmia occurring during the last ten years in the London, Middlesex, and St. George's hospitals,* 252 cases were males, and 69 females. This difference would have been less marked had cases arising from the puerperal state and the deaths from ovariotomy been included.

Table I.—Numbers of Patients at each Decade.

Sex.	1–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90	Total.
Male -	11	37	39	68	50	35	7	5	—	252
Female -	7	6	16	12	10	9	7	1	1	69
Total	18	43	55	80	60	44	14	6	1	321

No period of life is exempt from the development of these diseases. Among the youngest observed was that of a child aged five months, with suppuration of the knee joint and shoulder, the symptoms arising after

* Vide Appendix I.

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[illegible] Schweigger Jour. etc. Therefore (K. L. 39). Lowe and K[illegible] Ann. Chem. 81, 356) and Schlossberger (ibid. [illegible] were found only in the wing-cases of cockchafers (*Melolontha vulgaris*). The chitin was due to a constituent of the blood. The crusts are therefore a mixed tissue. Schmidt (Ann. Chem. Pharm. 91, 321) examined the shell of *Limulus Moluccanus*, Lam., Atherton saver. Astacus fluviatilis, Rondelet, Nephrops norvegicus, Linn., and found the elementary composition as above. Schlossberger (loc. cit., 227) examined the shell of *Palinurus vulgaris* and found in its chitin 6·4 per cent. N.

The shells of crustacea contain up to 65 per cent. of organic matter, the mineral constituents are mainly carbonate and phosphate of calcium.

The crab (*Cancer pagurus*) contains: organic matter 33·8 per cent.; calcic carbonate with little sodic chloride, iron, manganese and pigment 61·0 per cent.; calcic phosphate 5·2 per cent. (John, Chem. Schriften, 3, 49.)

Crayfish shells: organic matter 28·0 per cent.; calcic carbonate 59·0 per cent.; calcic phosphate 13·0 per cent. (Morat and Guillot, Ann. Chim. 71.)

Crayfish (*Astacus*): chitin 46·73 per cent.; ash 53·27 per cent. In the ash 40·00 per cent. calcic carbonate and 13·17 calcic phosphate. (K. Schmidt, loc. cit.)

Shell of crayfish: Organic matter 36·5 per cent.; calcic carbonate 56·9 per cent.; calcic phosphate 6·7 per cent. (Fremy, l.c.)

Nerve of crab: Organic matter and water 28·6 per cent.; sodium salts 1·5 per cent.; calcic carbonate 62·8 per cent.; calcic phosphate 6·0 per cent.; magnesic phosphate 1·0 per cent. (Goebel, Schweigger's Journ. 39, 440.) The same material found in the claws of crab: organic matter 17·15 per cent.; calcic carbonate 36 per cent.; calcic phosphate 14·06 per cent. In the teeth of the claws: organic matter 12·75 per cent.; calcic carbonate 68·25 per cent.; calcic phosphate 18·75 per cent. Shell of crab: organic matter 28·0 per cent.; calcic carbonate 62·50 per cent.; calcic phosphate 6·0 per cent. (Chevreul, Ann. [illegible] Nr. 15, p. 4, 124. Schweigger's Journ. 32, 495.)

Nerve of squilla mantis: chitin 62·84 per cent.; salts 13·17 (of these calcic phosphate formed 47·32, and carbonate 32·48.) (K. Schmidt, loc. cit.)

Claws of lobster: Organic matter and water 44·76 per cent.; sodium salts 1·5 per cent.; calcic carbonate 49·26 per cent.; calcic phosphate 3·22 per cent.; magnesic phosphate 1·26 per cent. (Chevreul, l.c.)

Claws of lobster: Organic matter 28·0 per cent.; calcic carbonate 40 per cent.; calcic phosphate 14·0 per cent. (Guillot, l.c.)

Shell of lobster: Organic matter 44·3 per cent.; calcic carbonate 49·0 per cent.; calcic phosphate 6·7 per cent. (Fremy, l.c.)

Summary of Historical Literature.—Chitin was first distinguished from bone substance by Odier in 1823, and was supposed by him to be free from nitrogen (Mem. Soc. d'Hist. Nat. de Paris, 1, 29). Lassaigne recognised that it named by him entomoderm, contained nitrogen (Compt. Rend., 10, 395; Journ. Chim. Med., 19, 379). It was next studied by Payen (Compt. Rend., 17, [illegible], and more intimately by K. Schmidt (Ann. Chem., 58, 29; Schlossberger Ann. Chem., 58, 105) contributed to its more accurate knowledge by defining the organic matter contained in some shells and in the bones of shells which had been confounded with chitin, as constituting it. Berthelot (Compt. Rend., 247, 727, and in detail N. Ann. Chim. Phys., 30) found that chitin on chemolysis with acid gave a kind of sugar. (Compt. Rend., 47, 1034; N. Ann. Chim. Phys., 58, 83) in connection endeavoured to find the amylum-like body in chitin which gave the Staedeler Ann. Pharm., 111, 21) also studied chitin, and gave a new formula different from that first given by Schmidt. G. Ledderhose (Zeitschr. Chem., 2, 1873) 213) found the sugar-like body to contain nitrogen, termed it glykosamine. He also proposed a new formula for chitin, which has been discussed in the foregoing.

No. 4.

Report on Pyæmia, Septicæmia, and Purulent Infection.

to the Pathological Society of London by the Committee pointed "to investigate the Nature, Causes, and Prevention of the Infective Diseases known as Pyæmia, Septicæmia, and Purulent Infection."

beg to submit the results of the investigation into the nature ses of those infective diseases known as pyæmia, septicæmia, rulent infection undertaken at the request of this Society in ice with an engagement entered into by the Society with the overnment Board.

plan of investigation followed by your Committee has been:

o collect statistical information with respect to the affections in question from nearly all the large London hospitals during the decennial period from 1869 to 1879, with a view of determining the history of the disease in the surgical wards of the metropolitan hospitals during that decennium.

The collation of 155 detailed reports of cases, with the object of attempting to classify them according to their clinical and pathological features, and of ascertaining how far these correspond with the diseases produced experimentally in animals.

An examination of a considerable number of microscopical preparations of the various organs of the body from typical cases, together with observations on the blood and urine, in some cases made during life, to illustrate the nature of the pathological changes in different stages of the various septic diseases.

PART I.

(1.) *Sex and Age.*—Out of 321 cases of pyæmia and septicæmia curring during the last ten years in the London, Middlesex, and t. George's hospitals,* 252 cases were males, and 69 females. This ifference would have been less marked had cases arising from the uerperal state and the deaths from ovariotomy been included.

Table I.—Numbers of Patients at each Decade.

Sex.	1–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90	Total.
Male -	11	37	39	68	50	35	7	5	—	252
Female -	7	6	16	12	10	9	7	1	1	69
Total	18	43	55	80	60	44	14	6	1	321

No period of life is exempt fr… t of t… ases.
Among the youngest pa… d fi… ith
suppuration of t… pte… ter

vaccination; and the oldest, a woman in her ninetieth year, with old pelvic abscess. The decade, however, which furnishes the largest number of cases is that between 30 and 40,—a circumstance naturally attributable to the greater exposure to accidents and injuries at that period of life among the working members of the community.

(2.) *Influence of Seasons and Meteorological Conditions.*—Table II. gives a return of the deaths from pyæmia originating in the surgical wards of eight large London hospitals during the decennium. The average mortality in each year amounted to 92·1; the highest death-rate occurred in 1874, when it reached 125, and the lowest in 1864, when it was 64. But as the returns for that year do not include those of University College, then the mortality in 1878, which does, must be taken as the lowest.

TABLE II.—DECENNIAL RETURN OF DEATHS FROM PYÆMIA IN 8 LONDON HOSPITALS.

Hospital.	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878
St. Bartholomew's	10	9	10	8	4	8	9	8	3	9
St. George's	11	15	11	7	11	8[1]	8	5	5	4
Guy's	21	23	28	19	29	30	38	26	23	13
London	10	7	17	22	19	29	27	16	21	12
Middlesex	4	6	10	7	8	9	3	5	5	1
Seamen's	4	5	9	8	5	10	7	6	4	2
St. Thomas	4	4	7	19[2]	16	13	16	12	22	12
University	—[3]	—[3]	12	8	7	18	11	10	5	14
TOTAL	64	69	104	98	99	125	119	88	88	67

[1] Hospital closed for three months from June to September.
[2] First year in new buildings.
[3] Returns not given.

On referring to this table it is interesting to observe that in the years of low mortality the death-rate is nearly in all cases low at all the hospitals, and in like manner the death-rate is high in the years of excessive mortality, pointing to a conclusion that the mortality is due in great measure to general as well as to local conditions. The years 1874 and 1875, in which the greatest number of deaths from pyæmia occurred, were ones of comparative high mortality for all diseases throughout the kingdom.* In the year 1874 the cases of erysipelas admitted into the London hospitals were especially numerous (Table IV.). Both years were characterised by atmospheric vicissitudes of opposite characters. In 1874† there was a remarkable deficiency in the rainfall during the whole year, the total being 5·4 inches below the average. The mean average temperature of January was remarkably high, being

* The deaths registered in England from diphtheria, erysipelas, and puerperal fever to every 1,000,000 persons living were exceptionally high in 1874 and 1875, as is seen by the appended table, taken from the Registrar-General's Returns:—

Disease.	1870	1871	1872	1873	1874	1875	1876	1877
Diphtheria	121	112	94	109	152	143	131	112
Erysipelas	96	99	78	88	144	128	97	86
Puerperal fever	67	65	61	75	132	105	72	59

† Remarks on the meteorology of 1874 and 1875; Thirty-seventh and Thirty eighth Annual Reports of the Registrar-General.

5°·4 above the average of 103 years. During February, and in the early part of March, the temperature was variable, there being 18 days when the temperature was below, and 20 days when it was above the average. On the 14th of March a spell of warm weather set in, which lasted to the end of April, the mean temperature of April being 50° F. On referring back 103 years there have been only five Aprils of so high a temperature. On the 1st of May a cold period set in, which continued without exception till the 21st. These three weeks of low temperature were very trying, following so immediately the heat of the preceding seven weeks. The temperature during the summer and autumn was variable, but the variations were not excessive. On the 21st November, however, a cold period set in, which lasted, with but few exceptions, till the end of the year. Since the year 1771 there have been only seven instances of so low a mean temperature as in December 1874. Unlike the preceding year, 1875 was characterised by its excessive rainfall. The excess, however, was not spread equally over the whole year, but occurred in its last half, the total fall in the six months ending 30th June being 1·0 in. below the average. In July, however, the fall became excessive, being 2·7 in. above the average. Back to 1815 there have only been four Julys with rainfalls so large. October was also an extremely wet month, and the other months of this half of the year, with the exception of August, had more than the average rainfall. Fog too prevailed on 152 days of this year. The vicissitudes of temperature were considerable. January was a warm month, the daily excess of temperature on 29 days over the average being as great as 10°, 11°, and 12°. Then set in bitterly cold weather, lasting all February and March, the wind remaining almost continuously E. during this period. April and May were variable, but the summer from June 11th to August 6th was cold and damp. September was warm, but October generally cold and wet. The weather again became very severe from the 20th of November to the 16th of December, with a very long continuance of E. wind. The atmospheric vicissitudes of these two years were certainly more severe than in other years of the decennial period. Table III. gives the mortality from pyæmia and septicæmia in each month in each year, taken from the reports of St. George's, the London, and University College Hospitals.*

TABLE III.—MONTHLY DEATH-RATE FROM PYÆMIA AT 3 LONDON HOSPITALS DURING DECENNIAL PERIOD.

Year.	Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
1869[1]	5	3	—	1	1	4	1	1	2	1	3	2
1870[1]	1	8	1	3	2	1	—	2	1	—	2	1
1871	4	8	5	2	2	3	7	1	—	2	3	—
1872	2	1	7	2	4	4	4	6	2	2	2	1
1873	6	3	7	5	6	—	2	—	1	2	3	1
1874	2	10	4	1	6	2	3	2	5	5	4	8
1875	—	7	7	3	3	5	2	3	3	3	6	3
1876	5	4	1	2	4	1	5	2	2	1	2	1
1877	1	4	5	3	2	2	1	1	3	4	3	1
1878	1	3	3	3	4	2	4	4	3	1	1	2
TOTAL	27	51	40	25	34	24	29	22	22	21	29	20

[1] Returns of University College Hospital not included in these years.

* Vide Appendix I.

The average monthly death-rate for the decennium stands at 28·5, but for the months of February and March the mortality rises to 51 and 40 respectively. May is the next fatal month, with a mortality of 34. The death-rate for these months is therefore considerably in excess of the other nine months of the year, and the question arises, does this excess, which seems pretty constant, bear any relationship to sanitary or meteorological conditions, such as distance from the period of annual hospital cleaning, or the necessity of having the ward windows closed for longer periods together during the usually prolonged wet and cold weather generally characteristic of February, and during the east winds which often prevail for many days together in March and May. Besides this, the vicissitudes of temperature in these months are usually great, and a return of cold after a few warm spring days is sure to be felt by the inmates of the hospital, and thus lead to undue closing of windows and other channels of ventilation.

(3.) *Relationship of Pyæmia to Erysipelas.*—Table IV. contrasts the deaths from pyæmia and septicæmia originating in the wards of St. Bartholomew's, St. George's, Middlesex, St. Thomas's, and University College Hospitals* with the cases of erysipelas under treatment at those hospitals in each year for nine years of the decennium.

TABLE IV.—RELATIONSHIP BETWEEN PYÆMIA AND ERYSIPELAS.

——	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878
Pyæmia - -	29	34	50	49	46	56	47	40	35	
Erysipelas - -	219	199	203	365	412	457	352	312	288	

No very certain relation seems to exist between pyæmia and erysipelas. The year 1874, it is true, was characterised by an excess of both forms of the disease, but 1871, which had the next highest mortality from pyæmia among these hospitals, suffered comparatively slightly from erysipelas.

(4.) *Duration of the Disease, and the frequency of its Occurrence after various Surgical Lesions.*—In 142 cases originating in St. Thomas's, St. George's, and University College Hospitals in which the date of the invasion was stated, the average stay in hospital from admission to onset of pyæmic symptoms was 20·2 days, and the period intervening between invasion and death in 153 cases from the same hospitals was 9·5 days.* In 768 cases taken from the records of St. George's, London, Middlesex, St. Thomas's, and University College Hospitals* the disease followed operations in 218, injuries in 120; it arose during the progress of some surgical disease in 268, and in 96 cases the disease either appeared due to a variety of conditions or no distinct cause was assigned for it. The particulars of the nature of the operation, injuries, diseases, &c. are given in Table V., arranged as to their occurrence at each of the hospitals from which the cases were taken.

* Vide Appendix I.

(5.) Table V.—Analysis of 768 cases of Pyæmia arising in six London Hospitals during Decennium.

Surgical Lesion.	St. George's.	Guy's.	London.	Middlesex.	St. Thomas's.	University College.	Total.	
Amputation of limbs	11	54	38	4	27	11	145	Operations 217
Amputation of breast	1	—[1]	3	1	3	—	8	
Removal of tumours	2	12	—	1	3	—	18	
Herniotomy	1	1	—	—	2	—	4	
Osteotomy	—	1	—	—	2	—	3	
Lithotomy	—	—	—	2	1	4	7	
Lithotrites	1	—	—	—	—	—	1	
Ligature of arteries and piles	4	2	3	1	2	—	12	
Tapping	—	—	—	1	2	2	5	
Incisions	—	—	—	1	2	—	3	
Trephining and fractured skull	5	—	1	1	—	4	11	
Galvano-cautery	—	—	—	—	1	—	1	
Compound fractures and dislocations	10	—	23	5	9	3	50	Injuries 120
Simple fractures	2	—[2]	12	—	1	3	18	
Lacerated wounds	3	22	16	5	3	1	12	
Contused wounds	—			—	1	1	30	
Urethral	7	34	2	2	7	14	66	Urethral 66
Diseases of bone	7	34[3]	17	1	5	8	72	Surgical diseases 268
Diseases of joints	6[3]	41[3]	17[3]	5[3]	24[3]	11[3]	104	
Abscess	10	5	11	6	11	7	50	
Ulcer	2	7	4	1	2	—	16	
Carbuncle	1	7	3	1	—	—	12	
Whitlow	—	—	—	—	—	2	2	
Scald or burn	—	2	1	—	—	—	3	
Cancer	—	—	3	3	—	1	7	
Bedsores	—	—	—	1	—	1	2	
Various	3	31[4]	16	2	6	5	63	
No cause stated	9	4	10	—	10	—	33	
Total	85	257	180	44	124	78	768	

[1] Cases of amputation of the breast classed with removal of tumours.
[2] Diseases of bone also include simple fractures.
[3] Diseases of joints include excisions.
[4] Among this class are 11 cases of idiopathic pyæmia and 1 case of exopthalmic goitre.

(6.) Before passing from this part of the subject we would observe that, considerable as the number of cases collected in these statistics may be, still, spread as they are over a number of years, they are insufficient to found definite conclusions upon, but may prove useful as suggesting points for future inquiry. They would, however, have been more conclusive than they are had the whole material of the London hospitals been available; but, unfortunately, owing to the absence of any uniform system of keeping the registers throughout the metropolitan hospitals, information given in some reports is not mentioned in others. It would be of immense value, especially in determining the etiology of diseases in points relating to their predisposing causes, if an uniform system of registration were adopted for all the metropolitan hospitals.

Report on Pyæmia, Septicæmia, and Purulent Infection.

PART II.—CLINICAL.

(7.) In this part of the inquiry it has been attempted by the collection and comparison of a considerable number of cases of septicæmia and pyæmia to ascertain whether these diseases can be divided into groups sharply defined from one another, and how far these groups correspond in their clinical features and naked eye pathological appearances to the diseases of the same kind artificially produced in animals. The question as to the presence or absence of bacteria in the blood and tissues, and the part that these organisms play in the causation of the disease, will be treated in the next section of the report, and has been as far as possible omitted in this.

(8.) *Septicæmia.*—At the outset of this portion of the inquiry we are met with the difficulty that the term septicæmia is employed by different authors with very different significance. Thus, Dr. Sanderson, speaking of septicæmia in his lectures delivered in connexion with the Brown Institution, says, "What I mean by septicæmia is a constitutional disease of limited duration produced by the entrance* into the blood stream of a certain quantity of septic material. It must, therefore, be regarded not so much as a disease as a complication, differing from pyæmia not only in the fact that it has no necessary connexion with any local process, either primary or secondary, but also in the important particular that it has no development. Pyæmia is a malignant process which goes on and on to its fatal end, but in the case of septicæmia, inasmuch as the poison which produces it has no tendency to multiply in the organism, there is no reason why the morbid process should not come to an end of itself unless either the original dose is fatal, or a second infection takes place from the same or another source." On the other hand, Davaine and those who have followed him describe as septicæmia every case in which death follows the entrance of putrid substances into the blood-stream, without the formation of local centres of inflammation or suppuration. Koch† has followed Davaine, and includes under the term septicæmia "all those cases of general infection from a wound in which no metastatic changes occur."

Birch-Hirschfield limits the term septicæmia much in the same way as Dr. Sanderson. He describes as septicæmia those cases in which the disease results merely from the absorption of the products of putrefaction, and regards it merely as a process of poisoning such as might arise from the injection of any other noxious chemical substance into the blood. Pyæmia, on the other hand, he considers a truly infective process, probably due to the entrance of specific organisms into the body. He would therefore include the second class of cases described by Koch as septicæmia under pyæmia. Hueter‡ defines septicæmia as a fever caused by the entrance into the circulation of the products of putrefaction from local centres of decomposition. He draws no clear distinction between an infective and a non-infective form, but the affection he describes as pyæmia simplex or pyæmia without metastasis seems to include many cases which Davaine, Koch, and others would include under septicæmia.

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* Lectures on the Infective Processes, delivered at the University of London, by J. Burdon Sanderson, M.D., LL.D., F.R.S. Brit. Med. Journ., Dec. 29, 1877.

† Untersuchungen über die Aetiologie der Wundinfections Krankheiten, von Dr. R. Koch, Leipzig, 1878.

‡ Handbuch der allgemeinen und Speciellen Chirurgie; Billroth and Pitha, 1872.

Billroth* defines septicæmia as "a for the most part acute general "affection which arises from the taking up of various kinds of putrid "substances into the blood, and it is believed that these putrid substances "so change the quality of the blood that it can no longer fulfil its "physiological functions." Billroth, like Hueter, does not draw any sharp distinction between an infective and a non-infective form. Many other observers have stated that the disease they speak of as septicæmia shows an increasing virulence when transmitted by inoculation from animal to animal, thus indicating a multiplication of the poison in the organism. Observations of this kind have been recorded by Davaine, Coze and Feltz, Hiller, and others.

(9.) Leaving the experimental side of the question and turning to the more purely clinical, we find that many authorities, especially in this country, recognise no essential difference between any forms of septicæmia and between septicæmia and pyæmia. Thus Mr. Savory, speaking in the debate on pyæmia at the Clinical Society in 1874, classes septicæmia and pyæmia together as but different degrees of effect of the same poison, septicæmia being more acute and terminating fatally before there has been time for the formation of secondary points of suppuration. Mr. Hulke, on the other hand, would clearly distinguish between pyæmia and septicæmia, and thinks a sharp line of definition should be drawn between the two. At the same time he thinks it may be difficult to do so in practice at the bedside, because these conditions may co-exist in the same patient.

If we refer to three of our chief text books of surgery we find that Mr. Erichsen makes some distinction between pyæmia and septicæmia, although not actually treating the two conditions as separate diseases. Mr. Bryant opens the chapter on traumatic fever, septicæmia, and pyæmia thus: "Inflammatory fever, surgical, suppurative, or traumatic "fever, septicæmia, ichoræmia, puerperal fever, and pyæmia may all "be considered as so many different names for and manifestations of one "condition—blood poisoning." Mr. Holmes also states that septicæmia and pyæmia are identical.

It is needless to quote any further to show that the term septicæmia as at present used cannot be said to have any definite meaning at all. It seems probable that at least two separate and distinct conditions exist to which the name septicæmia is commonly applied. These two forms of disease cannot be better illustrated than by referring to the observations on artificial infective diseases of wounds by Dr. Robert Koch.† His experiments were made upon the common mouse, and were as follows:—Five drops of putrid blood or meat infusion were injected under the skin of the back. Almost immediately the animal became restless, it ceased to eat. Soon its movements became weak and uncertain, the respiration was irregular and gasping, and in from four to eight hours death took place. Microscopic examination of the seat of injection showed no sign of inflammatory reaction. The internal organs were unchanged. No bacteria were to be found in the blood nor in the viscera, and a drop of blood taken from the heart and injected into another mouse produced no ill effects. It was evident here that no true infective process had been started; the animal died from the chemical poison just as is might have done from a dose of arsenic. Further experiments showed that the effect produced by the injection was proportional to the

* Billroth, Lectures on Surgical Pathology and Therapeutics; translated for the Sydenham Society, 1878.

† Op. cit.

dose, and that when a sufficiently small quantity was injected the animal usually escaped without any ill effects. A certain proportion of the mice experimented on, however, after about 24 hours of apparent immunity, began to show symptoms different from those just described, and quite as characteristic. The proportion of mice thus affected varied with the quantity of the putrid fluid injected. If one drop were used about one third of the experiments succeeded; if from one-tenth to one-twentieth of a drop only about one in ten or twelve. The earliest symptom in the successful cases was an increased secretion from the conjunctiva which finally stuck the eyelids together. At the same time the animal became weak, showed little tendency to move, but sat still, and with extended legs. It ceased to eat, the respiration became slower, and debility increased, and finally death occurred quietly at the end of 40 to 60 hours after the injection. On examining the body after death, a slight œdema was usually found at the seat of injection, all the internal organs being apparently unchanged, with the exception of considerable swelling of the spleen. The blood from the heart and the œdematous fluid from the seat of injection were found to possess the most intense infective properties. In many cases it was sufficient to scratch the ear or the root of the tail of another mouse with a scalpel dipped in the blood of the first in order to set up the same disease. Here then was set up an undoubtedly infective disease of the most virulent character, agreeing in many of its clinical features with what we know as septicæmia in man. Microscopic examination of the blood and internal organs of these mice showed, moreover, a peculiar small bacillus invariably present in enormous numbers. It is needless to go into further detail with regard to these most interesting observations. Sufficient has been given to indicate clearly the difference between mere poisoning from the chemical products of decomposition and the establishment of a true infective process. In the one case the poison does not multiply itself in the blood of the animal; the effects are purely proportional to the dose and the size and susceptibility of the animal experimented on; the blood after the death—merely containing the original dose of poison diluted—is incapable of setting up a similar intoxication in another animal. In the other case the poison has increased in the body of the affected animal, and the smallest drop of the blood or of the inflammatory exudation will start a similar process in another animal. The establishment of a clear distinction between these two conditions may serve to explain many of the apparent contradictions which exist in the multitudinous observations upon experimental septicæmia.

In order clearly to distinguish these two conditions from each other it would be, perhaps, better to employ the term used by some of the German writers, and to give to the former the name of septic or putrid intoxication, and to the latter septic or putrid infection.

(10.) With regard to septic intoxication we owe the chief part of our accurate knowledge to Bergmann and Panum and Billroth. These authors clearly showed that a substance could be extracted chemically from putrefying animal matter, which when injected into the blood-stream caused death with the ordinary symptoms of septicæmia. The exact nature of this substance is not known. Bergmann thought he discovered something definite, to which he gave the name of *sepsin*, but later observers have not proved the existence of this in putrefying animal fluids.

Our knowledge upon the subject of septic intoxication as derived from experiments upon animals may be briefly summed up, thus:—

The effect of the dose is proportional to the size and susceptibility of the animal experimented on. Carnivora are less susceptible than herbivora. Rabbits and guinea-pigs are extremely susceptible, dogs much less so. It is probable that the susceptibility of the human subject is greater than that of dogs; it is certainly much less than that of guinea-pigs or rabbits.

It is probable also that the susceptibility of the individual varies with the state of health at the time. The poison does not multiply in the body, and consequently the blood of an animal which has been killed by pure septic intoxication, containing as it does only the original dose diluted, will not set up a similar affection in another animal.

According to the observation of Koch, organisms are not found in the blood of animals suffering from septic intoxication. The symptoms produced by a fatal dose of the septic poison are—restlessness, muscular weakness gradually increasing until the animal falls down unable to stand; also vomiting and profuse diarrhœa, the fæces at first being loose and whitish grey in colour, and afterwards blood-stained or containing blood clots; the respirations and the heart's action become gradually more and more feeble until death ensues, sometimes preceded by cramps; the temperature at first rises some degrees, but before death often falls below normal.

The chief post-mortem appearances are as follows:—

The blood is dark in colour, and coagulates imperfectly. Petechiæ are found both beneath the pericardium and endocardium chiefly in the left ventricle, and also beneath the pleura. The mucous membrane of the stomach and intestines is intensely injected, and here and there are patches of extravasated blood. The epithelium has desquamated in patches or entirely. The spleen is dark in colour, engorged with blood, soft and often pulpy.

Dr. Sanderson summarises the post-mortem appearances thus:—
"First, there is an obvious tendency to congestion and capillary "hæmorrhage; and secondly, a localised congestion of the gastro-"intestinal mucous membrane, which is so intense that we are tempted, "with Bergmann, to speak of it as gastro-enteritis."

Microscopic examination of the blood shows that the corpuscles tend to aggregate themselves into clumps instead of rouleaux. The serum is also found to be stained with the blood pigment immediately after death, showing that the disintegration of the corpuscles has commenced during life.

The appearances above briefly noted have been described without any material variation by all authors from the time of Gaspard (1822) onwards. It must, however, be noted that Koch, in his experiments on mice, found the internal organs unaltered after death, caused by the injection of ten drops of putrid blood beneath the skin of the back.

If the dose used be not sufficient to cause death, the symptoms gradually subside, and the animal recovers more or less weakened by what it has gone through, but apparently without suffering any permanent injury.

As a result of the above observations, the theory has of late years been generally accepted that the fever accompanying large recent wounds, in which decomposing matter is in contact with the raw surface, is in great part or entirely due to the absorption of septic matter, and that this fever continues until the surface becomes covered by healthy granulation-tissue, which is believed to prevent further absorption, unless the septic fluids are pent up in contact with it at some degree of pressure. Accepting this theory as true, ordinary wound fever is merely septic intoxication in a very mild form, and it is only necessary

for the dose absorbed to be sufficient in quantity for fatal consequences to ensue. Septic intoxication is therefore of the commonest possible occurrence, as a complication of severe surgical injuries, but it is in so mild a form as to bear but little resemblance to that experimentally produced in animals.

(11.) It has been attempted in this part of the inquiry to ascertain, by collating and comparing fatal cases of so-called septicæmia, if anything at all comparable to experimental septic intoxication occurs in the human subject. The conditions under which such an accident can occur are not of frequent occurrence. In the first place there must be a sufficient quantity of septic matter to furnish the fatal dose in the human subject; and secondly, there must be conditions favourable to its rapid absorption. We have no definite knowledge of the quantity of putrid serum necessary to furnish a fatal dose in the human subject. In Mr. Savory's well-known experiments on blood-poisoning (St. Bartholomew's Hospital Reports, 1865), a putrid fluid (obtained by macerating cat's flesh in water) and putrid liquor amnii, were injected directly into the veins of the animals experimented on. The quantity required to prove fatal to a large cat or to a dog of from 12 to 15 lbs. seems to have been about two drachms or more. The effect of the poison being proportional to the bulk of the animal, this corresponds to about 2½ oz. in the human subject; but the susceptibility of man being supposed to be greater than that of the dog, the quantity required would be smaller and again the fluids, used containing less albumen than serum, would probably not present the poison in so concentrated a form. It must be acknowledged, therefore, that there are no definite data from which we can determine the fatal dose of putrefying serum or pus in the human subject, but the probability is that it would require from one to two ounces to be absorbed within a comparatively short time to procure anything analogous to the acute experimental septic intoxication of animals. In children a smaller quantity would be required. The conditions necessary for its rapid absorption could be found in all recent wounds, the raw surfaces of which before they are covered by healthy granulation-tissue form one of the most active absorbing surfaces conceivable. The absorbing power of the wound would of course vary with its extent. Absorption also takes place with great rapidity from serous or synovial cavities. From the cavities of acute abcesses absorption does not take place readily through the granulation-tissue forming the wall, unless the septic matter is pent up at some degree of pressure. The conditions therefore under which it is theoretically probable that acute septic intoxication might occur in the human subject are large and irregular wounds, such as those resulting from compound fractures of the bones of a limb; hollow wounds, such as those left after the removal of tumours; wounds of joints; wounds involving the pleura and peritoneum, and large abscesses opening externally by an insufficient aperture. The accumulation of septic matter in the uterus after labour, in contact with raw surface left by the separation of the placenta, would also present the conditions favourable to acute septic intoxication. In the present day, when the necessity of thorough drainage of wounds is so fully understood, and the means at the surgeon's command for carrying it out are so efficient, it can only be under peculiar circumstances that a sufficient quantity of putrid serum or pus to yield the fatal dose of the septic poison is allowed to accumulate in a wound. Moreover the antiseptic treatment of wounds now—so largely adopted—by preventing decomposition of course renders septic intoxication impossible. Ovariotomy would seem to furnish conditions most favourable to septic intoxication, a large proportion of the deaths

occurring in the first forty-eight hours have always been attributed to it. The proportion of fatal cases from this cause has, however, of late been greatly diminished by drainage, and more especially by the employment of the antiseptic treatment.

The evidence therefore to be looked for as an indication of fatal acute septic intoxication in the human subject would be—

1. The presence of a sufficient quantity of septic matter to yield the necessary dose of the septic poison.
2. The invasion of the symptoms at a time when decomposition of the discharges has commenced, and before the surface of the wound has been covered by granulations.
3. High fever rapidly followed by great depression.
4. Post-mortem appearances indicative of a "tendency to congestion and capillary hæmorrhage."
5. Accepting Koch's observations as correct, organisms would not be found in blood during life or immediately after death.

(12.) Of the 155* cases collected, 28 died without the formation of secondary centres of inflammation, and consequently would be classed by most authors as cases of septicæmia. In a great majority of these cases the conditions were such that it was possible for a fatal dose of septic matter to accumulate in the wound. Of these 28 cases the available notes only enabled us to ascertain with any degree of accuracy the time at which the secondary symptoms commenced in 14. In seven of these 14, or exactly one half, the symptoms commenced on the second day (cases 1, 3, 5, 12, 16, 18, 19, Appendix II., Table I.); in two they commenced on the third day (cases 6 and 10*), and in the rest at various periods up to three weeks. Of the seven cases in which the symptoms commenced on the second day one (case 5*) presented features quite distinct from the rest; the patient had repeated rigors and died on the twenty-second day. The remaining six died within a few days of the commencement of the symptoms. In four of these six cases (cases 3, 12, 16, and 19*) there was diffuse cellulitis spreading from the local injury, and consequently the suspicion is raised that some specific infective inflammation might have attacked the wound. In one of these (case 3) bacteria were observed in the blood during life, and there was strong suspicion of infection from another. In another (case 12) the wound was treated antiseptically, and there was no odour of decomposition in the discharges at the time of death. These cases must, therefore, be excluded as being probably due to a true infective process and not to pure septic intoxication. Only two cases, therefore, are left in which there is presumptive evidence that the patient died directly from the toxic effects of the products of decomposition. In the first (case 1) a large tumour was removed from below the lower jaw, and the cavity, apparently from want of efficient drainage, became distended with foul discharges. The patient lost but little blood at the operation. On the second day she was very weak and ill with an intermittent pulse. On the third day the distension of the wound was discovered and relieved, but in the afternoon of the same day there was severe dyspnœa and she died in the same evening. There were no rigors and no vomiting or diarrhœa. The patient was 74 years of age, and consequently might be supposed to be not very capable of withstanding the effect of the poison. There was no record of the temperature. At the post-mortem examination, marked post-mortem staining of the endocardium was observed. The liver and spleen were pale and soft. In the second case (case 19)'

* See Appendix II. for the classification of the 155 cases.

the patient was admitted into the hospital with a large abscess of the bursa patellæ. This was opened. The next day she became feverish and delirious, and rapidly sank and died two days afterwards. The temperature rose to 104·4 on the first day; pulse 150. The post-mortem examination showed congestion of the lungs and kidneys. Liver soft and oily. Nothing abnormal noticed in the spleen. Neither of these cases occurred during the course of the present inquiry, and consequently we are not able to report any examination of the blood for bacteria. It must be confessed that the evidence is by no means conclusive as to the nature of the affection. The cases collected being from general hospitals, they include none of ovariotomy, or perhaps more specimens of unmistakeable septic intoxication might have been obtained.

(13.) *Septic Infection without Metastatic Inflammation.*—By this is meant a true infective process started in the organism by the introduction of some virus developed in decomposing organic matter. The poison multiplies itself in the organism, and its effects are not directly proportional to the dose. Of the exact nature of the poison nothing is as yet definitely known. The observations of Koch, before quoted, would favour the view that the poison is in reality a microscopic organism which multiplies in the blood, and that this organism is essential to the production of the disease, just as the bacillus anthracis has been proved to be essential to the production of splenic fever. On the other hand, Hiller asserts that by the injection of putrid fluid which had been proved to be free from bacteria, he succeeded in inducing a fatal disease, in which the blood of the affected animal was capable of communicating a similar affection, and that by transference from one individual to another the poison showed a progressively increasing virulence, thus indicating a multiplication of the poison in the blood of the affected animal. If this be true, it would suggest that the poison was of the nature of an unorganised ferment.

(14.) As most authors have at present drawn no clear distinction between the purely chemical poisoning by septic fluid and the infective process, it is somewhat difficult to gather what are the morbid appearances seen after death in the latter condition. They seem, however, to be chiefly congestions of internal organs, especially of the spleen, with subserous and submucous petechiæ. The symptoms during life are fever with progressive emaciation and exhaustion. The development of the disease is slower and its course less rapid than in acute septic intoxication.

The conditions which in man would lead to the suspicion that the fatal result was due to the development of a poison multiplying in the blood, and not to the simple toxic effects of the products of decomposition, would be the following:—

1. The size of the wound being such as to render it impossible for the necessary fatal dose of septic matter to be formed in it;
2. Evidence of infection from one patient to another; and
3. If Koch's observations are correct, the presence of large numbers of bacteria in the blood during life or immediately after death.

Of course septic infection could originate from a large wound as well as from a small one, but such cases would in all probability be complicated by more or less septic intoxication, and the evidence as to the nature of the affection would therefore be correspondingly confused. It is also possible to suppose that a poison of such intense virulence might be developed in the decomposing discharges of a wound that an infinitesimal dose might be sufficient, if absorbed, to cause death without

any process of multiplication in the body of the affected animal. If so, however, the poison would become more and more feebly infective by inoculation of the blood of one animal to another, a condition the reverse of which has been observed in experimental observations on septic infection in animals. As to the evidence of infection from patient to patient, this cannot be considered necessary for every case of true septic infection, for the observations of all experimenters have shown that a truly infective process can be originated in an animal by the subcutaneous injection of putrid blood or other organic matter, but it is to a certain extent an accidental occurrence, or rather depends upon conditions not as yet accurately determined. When, however, the infective process is once started it may be transmitted from animal to animal with certainty. Evidence of infection, therefore, from patient to patient would indicate the existence of a true infective process, but a want of such evidence would not prove the contrary.

With regard to the necessary existence of microscopic organisms in the blood in septic infection we have the evidence of Koch, Davaine, Burdon Sanderson, Birch-Hirschfeld, and many others, that such is the case, and on the opposite side we have that of A. Hiller, which is supported by the great authority of Billroth.

(15.) Of the 28 cases collected, in which death occurred without the formation of secondary inflammations, only two arose from wounds so small as to exclude the possibility of sufficient septic matter being present to yield a directly fatal dose. The first of these (case 10) occurred in an old woman. A small epithelioma of the tongue was ligatured. The whole area included was not larger than a shilling. On the third day there was some elevation of temperature; this continued, and on the sixth day there was slight hæmorrhage from the raw surface. On the following day she was unconscious, and patches of congestion appeared on the skin. The skin was sallow in tint, and there was some inflammation of the conjunctiva on one side. There was great prostration. She died nine days after the operation and five after the commencement of the symptoms. The post-mortem examination showed a foul sloughy wound. Subserous petechiæ in pericardium and spleen. Swollen almost diffluent spleen. Swollen and blood-stained liver and kidneys. Great congestion of the lungs. The second case (case 23) arose in a dissecting-room porter. The primary affection was merely a little suppuration beneath the nails of the thumb and middle finger. The clinical features of the case could not be obtained. He was only one day in hospital. The blood was dark and fluid, there was congestion of the lungs, slightly soft and greasy spleen, swollen liver and kidneys, no petechiæ. The blood is stated to have contained no bacteria.

In two of the remaining cases (cases 3 and 4) bacteria were observed in the blood during life. One case of great interest unfortunately occurred during the absence from town of the member of the committee whose duty it would have been to make a detailed inquiry into the symptoms and nature. It was an excision of the knee performed with all antiseptic precautions, and to the time of death there was no odour of decomposition in the discharges (case 12). This case is the only one of the 28 in which it is not certain that there was decomposing animal matter in contact with the raw surface of the wound.

(16.) To sum up, therefore, the results of the comparison of the clinical and coarse pathological conditions noted in the 28 cases of so-called septicæmia, it may be said that in two there is strong presumptive evidence that death resulted directly from the toxic action of the products of decomposition developed in the wound at the primary seat of disease, and in two more of the existence of a true infective

process. In the remaining 24 cases, the evidence is not sufficient to form any definite conclusion as to the nature of the process. Death may have resulted from the prolonged absorption of the septic poison, leading to gradual death from exhaustion, or to the effect of a true infective process, or to both conditions combined.

The evidence of infection from patient to patient was not sufficiently definite in any case to form any conclusion from it.

Taking this group of cases as a whole, *i.e.*, those cases in which the post-mortem examination revealed no secondary or metastatic inflammation, the following were the chief symptoms and pathological appearances observed. The clinical history was only obtainable in 21 cases.

As it has been stated by some authorities that septicæmia differs from pyæmia only in time, and that if the patient survived sufficiently long, secondary inflammations would ultimately form, the duration of the cases has been calculated out, whenever the period of invasion was sufficiently accurately marked, with the following result:

About 2 days	-	2 cases.
„ 3 „	-	1 case.
„ 4 „	-	7 cases.
„ 5 „	-	1 case.
„ 7 „	-	2 cases.
„ 8 „	-	2 „
„ 10 „	-	1 case.
„ 14 „	-	1 „
„ 19 „	-	1 „
„ 22 „	-	1 „
Total	-	19 cases.

Some of these certainly survived sufficiently long for abscesses to form if it was in the nature of the case that such a thing should happen.

In eight of the cases in which the clinical history was obtainable rigors occurred, and in all but two of these they were repeated more than once. In one case (No. 5) in which the rigors were repeated no less than nine times, the patient survived to the 22nd day. It is evident, therefore, that nothing can be predicted as to the occurrence of internal inflammations or suppurations from the presence or absence of rigors; and also that the theory that the rigors are due to the commencement of fresh centres of secondary inflammation is untenable:—

Delirium	is noted in -	11 cases.
Vomiting	„ -	4 „
Diarrhœa	„ -	3 „
Dyspnœa	„ -	4 „
Icteric tint of skin	„ -	6 „

In the post-mortem examination of the 28 cases the following pathological appearances are noted:—

Congestion of lungs in -	23 cases.
Swollen liver -	14 „
Swollen spleen -	23 „
Congested and swollen kidneys -	16 „
Congestion of mucous membranes of intestines	1 case.
Subserous petechiæ -	2 cases.
Early decomposition -	3 „
Fluid blood -	2 „
Marked post-mortem staining -	7 „

(17.) The result of the whole of this part of the clinical inquiry cannot be said to do much more than show the necessity of further investigation in order to ascertain whether any distinct separation can be made clinically, as well as experimentally, between septic intoxication and septic infection. Septic intoxication is a condition which may occur whenever the discharges from a wound are allowed to accumulate in sufficient quantity to decompose. Theoretically, it should be absolutely preventable, and when it does occur does not necessarily give rise to anything which may be communicated from patient to patient. In septic infection, on the other hand, if we can draw any conclusion from the experiments upon animals, a virulent poison is produced which may be communicated from patient to patient with the most fatal consequences. It is, therefore, of the utmost importance to be able, if possible, to distinguish these conditions from each other.

(18.) *Pyæmia.*—The same difficulty arises with respect to pyæmia as was noted at the commencement of the part of the report referring to septicæmia. The term pyæmia is used by different authors with very different meaning. We have before alluded, in speaking of septicæmia, to the various uses of the term. The most common definition of pyæmia is no doubt that adopted by the College of Physicians in the "Nomenclature of Diseases." It is as follows:—"A febrile affection, resulting in the formation of abscesses in the viscera and other parts." This definition excludes all those cases which have just been described under septic infection. Birch Hirschfield includes under the name pyæmia all cases in which any general infective process is set up as a secondary consequence of a wound. Other authors seem still to be more or less wedded to the theory from which the word was originally derived. Thus, Hueter says, "Pyæmic fever is developed by the "absorption of the constituents of pus into the blood; the absorption "may be direct into the circulation of the fluids of the body (blood "and lymph), or it may take place after the previous absorption "of the constituents of the pus into venous thrombi, pieces of which "enter the circulation serving as carriers of the pus constituents." Billroth, in almost the same words, says, "Pyæmia is a disease which "we believe to arise from the taking up of pus and of the constituent "parts of pus into the blood. It stands in the same relation to simple "inflammatory fever and secondary fever as septicæmia does to simple "primary wound fever."

Koch, as before stated, uses the term pyæmia merely to denote a general affection accompanied by metastatic inflammations and suppurations. He says "the names pyæmia and septicæmia no longer express "what one originally designated by them,—for pyæmia does not arise, "as was formerly supposed, from the entrance of pus into the blood-"vessels, and septicæmia is not a putrefaction of the living blood. "They have finally only remained in use as general names for a number "of symptoms which most probably belong to a series of different "diseases."

It is needless to quote further in illustration of the various ways in which the term "pyæmia" is used. Although it would be much better if the word were entirely discarded and some other found in its place, it is very difficult to suggest any that would be free from objection, and as in the sense in which it is defined in the Nomenclature of Diseases of the College of Physicians, its clinical meaning is fully understood, no error is likely to arise from continuing to employ it.

The chief experiments with reference to pyæmia may be very briefly summed up as follows. It has been clearly shown by Dupuytren, Boyer, Sedillot, Virchow, O. Weber, Billroth, and many others, that

the injection of healthy pus free from solid particles visible to the naked eye does not cause any disease resembling pyæmia in any respect. Febrile disturbance of short duration is the only effect produced. If the pus be putrid and at the same time free from solid particles of any size, a fatal affection unaccompanied by metastatic inflammations or suppurations results from its injection into the blood stream.

No observer has as yet succeeded in inducing in animals a fatal infective process accompanied by metastatic inflammations and suppurations, by inoculation or injection of the blood and discharges obtained from a case of pyæmia in the human subject.

It has been demonstrated by Virchow, Sedillot, and by many others, that the introduction into the blood stream of solid particles which are themselves of an irritating nature, or are impregnated with the products of decomposition or of unhealthy inflammation, will cause the formation of metastatic abscesses, and O. Weber has shown that these solid particles may pass through lungs and lodge in other organs, thus causing similar results wherever they are arrested.

The nearest approach to the artificial production in animals of a disease resembling pyæmia in man has been made by Koch in some experiments recorded in the work before alluded to. The animals experimented on were rabbits. After failing several times to induce a general infective process with putrid blood, Koch tried other fluids with the same intention. The one with which he succeeded was prepared by macerating a piece of the skin of a mouse, one centimeter square, for two days in three grms. of distilled water. Ten drops of this fluid was injected beneath the skin of the back. The animal at first seemed to suffer but little. After 105 hours, however, it died. The examination after death showed a diffuse suppurative inflammation of the subcutaneous tissue extending from the back to the linea alba. In the belly the inflammation extended through the abdominal walls to the peritoneum. There was some peritonitis. The liver was mottled with grey patches, wedge-shaped on section. In the lungs were numerous dark red airless patches. Beyond these there were no other changes. The blood of this animal injected into others produced an essentially similar condition. Microscopic examination of the organs showed large numbers of micrococci, mostly single or in pairs. They measured about $0{\cdot}25\ \mu$ in diameter. In many parts they were seen in masses adhering to the walls of the capillaries, and enclosing some red corpuscles among them. Koch thinks that these accumulations lead to capillary thrombosis in many places, especially in the glomeruli of the kidneys. He suggests that such thrombi may possibly get washed out into the blood stream, and set up secondary abscesses. These observations have not, so far as we know, been at present confirmed. They are quoted here at length because of their novelty, and because, if confirmed, they will go far towards explaining the occurrence of the secondary abscesses in those cases of pyæmia in which no source of embolism can be detected by the naked eye.

(19.) For the purpose of grouping and comparing the cases of pyæmia which have been collected, the following classification has been adopted :—

I. Cases of pyæmia secondary to an open wound, in which there is no evidence that the secondary inflammations are due to embolism.

(*a.*) Cases in which visceral abscesses are wanting.

(*b.*) Cases in which visceral abscesses are present, but no softening thrombus is found at the primary seat of the disease.

II. Cases in which thrombosis has occurred apparently as one of the secondary affections.

III. Cases in which thrombosis and softening of the thrombus have occurred without giving rise to embolisms or secondary abscesses.

IV. Cases in which thrombosis and softening of the thrombus have occurred with secondary abscesses in other viscera, but not in the lungs.

V. Cases in which venous thrombosis with softening of the thrombus has occurred with abscesses in the lungs and in other viscera.

VI. Cases in which venous thrombosis and softening of the thrombus have occurred with embolic abscesses in the lungs only.

VII. Cases of pyæmia secondary to infective periostitis.

VIII. Cases of pyæmia secondary to infective endocarditis.

IX. Infective myositis.

X. Spontaneous septicæmia and pyæmia.

Group I.—Cases of Pyæmia in which there is no Evidence of Embolism.

(*a.*) *Without Visceral Abscesses.*

(20.) Twenty-four* of the 155 cases collected belong to this group. The local inflammations were distributed as follows:—

Joints only	8
Joints and subcutaneous tissue	3
Subcutaneous or inter-muscular tissue only	4
Inflammation of serous membranes only,	
Pleurisy	2
Peritonitis	1
Meningitis	2
Meningitis and pericarditis	1
Muscular substance of the heart (diffuse)	1
Muscular substance of the heart (diffuse), and areolar tissue beneath fascia lata	1
Suppuration of simple fractures and bruises	1
	24

The primary diseases or injuries may be classed as follows:—

Injuries or diseases affecting the genito-urinary tract	7
Diseases or injuries affecting the soft parts only, other than urinary organs	6
Amputations	5
Diseases of joints	3
Compound fractures	3
	24

In 19 of the cases the clinical history was obtainable, as well as the post-mortem appearances. In 15 of these the time of invasion after the commencement of the primary disease or injury could be ascertained

* Appendix II., Table II., Group I. (*a*).

with tolerable certainty. Ten commenced in the first week, two in the second, and the remaining three on the 20th, 23rd, and 31st days respectively (cases 1, 6, and 3).

The duration of the cases before the fatal termination was very various. It is tolerably accurately recorded in 18 cases. Of these, five died in the first week, five in the second, four in the third, and the remaining four survived to the 30th, 49th, 52nd, and 62nd days (cases 12, 5, 18, 10).

The following were the most prominent symptoms. Amongst the 19 cases fully reported, rigors occurred in 11 and were absent in eight. They were present in all the cases arising from diseases of the genito-urinary organs. They were present only three times amongst the nine cases who died during the first two weeks, but amongst the eight who survived that period they were only wanting once. Profuse sweating without the occurrence of rigors was noted in four cases. The temperature in every case was high, and when not complicated by rigors was tolerably regular. Diarrhœa was a prominent symptom in five cases, vomiting in six. In only one case are vomiting and diarrhœa noted as occurring together. Delirium was present in 8 cases.

The post-mortem appearances, in addition to the local secondary inflammations before noted, were in many cases those changes common to all forms of blood poisoning. In the 24 cases the following are noted:—

Swollen spleen	9 times.
Congested lungs	10 „
Swollen liver	6 „
Cloudy swelling of kidney	14 „

On comparing these figures with those given for septic intoxication and septic infection without metastasis, it will be observed that internal congestions are of much less frequent occurrence than in those affections.

In three of these cases the blood was examined during life for bacteria, but nothing was found (cases 5, 13, 14). The viscera were microscopically examined in four cases (cases 5, 13, 14, 19). In only one were any micrococci observed, and then only one colony was found after a very prolonged search, in the lymphatic gland nearest to a lithotomy wound.

Case 18 has been included in this group, although in some respects it differs from the rest. The blood poisoning arose from a compound fracture of the lower jaw, but the suppurations which followed were exclusively at the seats of various subcutaneous injuries which at first seemed progressing favourably. The case is especially interesting in connexion with the experiments of Professor Chauveau,* quoted by Professor Burdon Sanderson in his lectures on the infective processes of disease. These showed that a diffuse inflammation could be started in the seat of a subcutaneous injury by the injection into the blood stream of a dose of septic matter shortly after the occurrence of the injury. (Suppuration occurring at the seat of a subcutaneous injury is also illustrated by Case 9, Group III., Appendix II.)

The two cases of myocarditis (cases 16 and 17) were included in this group because the inflammation was general and diffuse, and nowhere had it given rise to localised collections of pus.

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* Étude experimentale sur les phénomènes de mortification et de putréfaction qui présent dans l'organisme animal vivant. Bull. de l'Acad. de Méd., 1873.

The comparatively large proportion of these cases that arose from affections of the genito-urinary system is a point of considerable interest, but the number of cases is so small that it may be merely the result of chance.

As a general summary of these 24 cases it may be said that the disease set up is a condition of blood-poisoning leading to a tendency to suppurative inflammation of the serous and synovial cavities and diffuse inflammation of tissues, unconnected in any way with embolism. The extremely chronic course of some of the cases is accounted for by the secondary inflammations not attacking vital organs. In all except some of the urinary cases, in which it was impossible, from the nature of the wound, to ascertain its exact condition, the discharges from the primary source of infection were in a state of putrefaction.

The cases collected being only those which terminated fatally, there is no evidence here given as to the mortality from this form of pyæmia, but it is a well-known fact that recovery is by no means uncommon.

(*b.*) *Cases in which Visceral Abscesses are present, but in which no evidence exists of Thrombosis and of disintegration of the Thrombus with Embolism.*

(21.) Of this form 28† cases have been collected, 16 with clinical features and history, and 12 with the post-mortem examinations only. The secondary inflammations and suppurations were distributed as follows:—

Lungs and subcutaneous tissue	3
Lungs and joints	2
Lungs, liver, joints, and subcutaneous tissue	1
Lungs and liver	3
Lungs, liver, and spleen	1
Lungs, liver, and kidney	1
Lung on one side, pleurisy on the other	1
Lungs only	8
Lungs and brain	2
Lungs and parotid gland	1
Spleen and kidney	1
Spleen only	1
Brain only	1
Liver, spleen, kidney, heart, pericardium, pleura, and joints	1
Subcutaneous tissue and pericardium	1
	28

The primary diseases may be classed as follows:—

Injuries or diseases of urinary organs	4
Diseases of the soft parts	10
Amputations	4
Diseases of and operations on joints	5
Compound fractures	1
Disease of bones	2
Ulceration of intestines	1
Doubtful	1
	28

* Appendix II., Table III., Group I. (*b.*)

The date of the invasion of the secondary disease after the commencement of the primary affection could not be ascertained accurately in more than 10 cases. In seven it was under five days; in the remaining three cases on the 8th, 14th, and 21st day. The character of the cases after the invasion could be tolerably accurately ascertained in 13 cases. Four died in the first week, six in the second, and the remaining three in the third. In none was the case prolonged as in the last group.

The chief symptoms are tolerably accurately reported in 15 cases. Rigors occurred in 12 of these and were absent in three. In both the urinary cases they were present. As in the previous group, the cases in which they were absent were amongst the most acute. The temperature was high and irregular, profuse sweating is mentioned as a prominent symptom in three, diarrhœa in four, vomiting in eight, delirium in five, jaundice in three, in all of which the liver was the seat of secondary abscesses. Rapid respiration is noted in two cases; a pustular eruption on the skin in one (case 12).

In addition to the metastatic inflammations before mentioned the following post-mortem appearances were noted: congestion of the lungs in 8, swollen liver in 5, swollen spleen in 19, swollen kidneys in 13. Although in all these cases no softening thrombus was detected in the vein which would suggest embolism as a cause for the secondary suppurations, it is quite possible that it may have existed and escaped observation. Thus, for example, of the eight cases in which abscesses were found in the lungs only, two were urinary cases, in which it is always difficult to trace the veins, one arose from diffuse cellulitis and incisions in the neck, one from a foul wound in the thigh, one from sloughy abscesses in the forearm and about the knee, and one from a foul abscess round the rectum; in one the limb was amputated after the first rigor and no record is given of the vein in the part removed, and in one the disease followed castration for tubercular testicle. The post-mortem notes state that the cut section of the cord exuded pus from minute abscesses. Possibly these were connected with the veins. In all these cases thrombosis might have been overlooked. A comparison of the post-mortem appearances of this group with those in which thrombosis was noted will be made after the summary of the latter.

Group II.—Cases in which Thrombosis occurred apparently as a Secondary Effect of Blood poisoning.

(22.) These six cases* are separated from the rest, not on account of any supposed difference in nature, but because they show that thrombosis of the veins at a part of the body distant from the primary disease, with softening of the thrombus and embolism, may occur as a part of the secondary manifestations of pyæmia. They suggest that if this may occur at a distance from the primary affection, possibly when the same changes are found at the primary seat of the disease, they may be due to an antecedent condition of blood-poisoning.

The six cases were as follows:—

Case 1.—Thrombosis, with softening of the clot and suppuration round the popliteal vein following amputation of the hand. One embolic abscess in the lung.

Case 2.—Thrombosis and softening of the clot in the veins of the leg secondary to old empyema with an external opening. Secondary abscesses in lungs and liver, and brain. Duration of case seven days.

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* Appendix II. Table IV., Group II.

Case 3.—Thrombosis and softening of the clot in the veins of the arm and forearm, five days after a compound fracture of the lower jaw. One small infarct in the lung; a little lymph on the surface of the brain. Duration of the case five days.

Case 4.—Thrombosis and softening of the clot in veins of the leg after removal of a small tumour from the tongue. Swelling of spleen, liver, and kidneys, and congestion of the lungs. Duration of the case about 16 days.

Case 5.—Thrombosis without softening of the clot in the veins of the leg after external urethrotomy. Congestion of the lungs and pulpy spleen. Duration of case 11 days.

Case 6.—Was a scirrhus of breast with a foul abscess in the axilla. The short saphenous vein was full of purulent fluid. Pus in the joints.

In Case 3 a microscopic examination was made of the organs after death. Micrococci were found in great abundance in the various organs. In the suprarenal capsules the blood-vessels in the medullary part were plugged with coagula containing groups of micrococci.

This observation is of great interest when taken in conjunction with Koch's experiments on artificial pyæmia in rabbits, before quoted, in which he is led to the conclusion that accumulations of micrococci in the capilleries may lead to thrombosis.

Group III.—Cases in which, although Venous Thrombosis and Softening of the Thrombus occurred, Death took place without the occurrence of Secondary Inflammations.

(23.) This small group of four* cases has been separated from the others as illustrating the fact that though thrombosis and softening of the thrombus may be present they may take no immediate part in the production of the fatal result.

Case 1.—Was a large psoas and iliac abscess opening on the thigh. The femoral vein was plugged with softening clot.

Case 2.—The symptoms commenced four days after an amputation of the thigh. The veins were swollen and filled with very friable clot.

Case 3.—Arose after excision of a large part of a varicose vein in the leg. The vein above the part removed contained a softening clot.

Case 4.—Followed retention of the placenta after abortion. The veins of the pelvis contained puriform clot.

In each of these cases the post-mortem appearances were those of "septicæmia." In all four the patient died within a short time of the invasion of the secondary disease. Had they lived longer, in all probability embolism would have occurred, and the cases would have presented the ordinary post-mortem appearances of embolic pyæmia. The last group of cases apparently showed that venous thrombosis and softening of the thrombus may be a result of a condition of blood-poisoning. In the present group the blood-poisoning seems to have preceded the thrombosis, and may possibly have been a cause of it. On the other hand, the co-existence of venous thrombosis and blood-poisoning in the same case may have been merely accidental. There is no evidence definitely to prove the relation of the two conditions to each other, one explanation being as plausible as the other.

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* Vide Appendix II., Table V., Group III.

GROUP IV.—CASES IN WHICH THROMBOSIS OCCURRED IN THE VEINS OF THE PRIMARY SEAT OF DISEASE, WITH NO SECONDARY ABSCESSES IN THE LUNGS, BUT INFLAMMATIONS AND SUPPURATIONS IN VARIOUS OTHER PARTS.

(24.) Of the 47* cases collected in which venous thrombosis was observed at the primary seat of disease, abscesses were not found in the lungs in nine. In these cases the secondary inflammations were distributed as follows:—

Joints and muscles	2
Joints only	4
Liver, spleen, endocardium	1
Liver only	1
Liver and spleen	1
	9

In the case in which the liver and spleen and pericardium were affected, the primary disease was ulceration of the gall bladder, and phlebitis of the portal vein (Case 6). In one in which the liver only was affected the primary disease was pericæcal ulcers (Case 5). These two may be explained as being embolism affecting the portal system. In four more (Cases 1, 3, 8, 9) the absence of embolism may be accounted for by the fact, that the softened thrombus was shut off from the blood stream by a barrier of unsoftened clot. In each of these cases the joints were suppurating and visceral abscesses were wanting. In one there was also pneumonia, but no signs of embolism. These would therefore seem to resemble closely the last group of cases, the thrombus, although occurring at the seat of the primary disease, being possibly determined by blood-condition which killed the patient. On the other hand they might be explained as being an accidental co-existence of a condition of blood-poisoning similar to that occurring in the cases included in Group 1 (*a.*) with thrombosis. In the three remaining cases (Cases 2, 4, 7) the joints only were affected in one, joints and muscles in one, and the liver and spleen in the third. In the first two of these cases it is difficult to understand how the patients escaped embolism of the lungs, as there seems to have been no barrier of clot as in the four cases just mentioned—with which they must be grouped. In Case 7 it is probable that the detritus of the clot was so fine that (as O. Weber has shown to be possible) it passed the lungs and lodged in other parts, as the liver and spleen presented distinct infarcts.

In this group, as in the others, the tendency for pyæmia secondary to diseases of the urinary organs to affect the joints is well shown. In each of the three urinary cases (Cases 2, 3, and 4) the joints were affected.

GROUP V.—CASES IN WHICH THROMBOSIS AND SOFTENING OF THE CLOT OCCURRED WITH ABSCESSES IN THE LUNGS AND IN OTHER VISCERA AND TISSUES.

(25.) In 13† cases of the 47 in which there was thrombosis of the veins leading from primary seat of disease, the secondary abscesses

* Appendix II., Table VI., Group IV.
† Appendix II., Table VII., Group V.

occurred in the lungs, but were not limited to them. They were distributed as follows:—

Lungs and spleen	3
Lungs and liver	2
Lungs, kidney, and heart	1
Lungs, spleen, and kidney	1
Lungs and joints	1
Lungs and subcutaneous tissue	1
Lungs and brain	2
Lungs, spleen, brain, kidney, subcutaneous tissue, and heart	1
Lungs and various parts injured subcutaneously	1
	13

The primary affection in these cases may be subdivided thus:—

Compound fracture	1
Amputations	5
Diseases of bone	2
„ joints	1
„ intestinal canal	1
„ urinary organs	2
Subcutaneous tissue	1
	13

The time of the invasion of the symptoms after the commencement of the primary disease could only be ascertained in five cases. In four it began in the second week, and in one in the fourth. The duration of the case after invasion was ascertained in nine, and was as follows:— One 2 days, one 4 days, one 5 days, three 7 days, one 11, one 12, and one 20. In these nine cases rigors were wanting in three, and in only two (that which survived 20 days, and one other, Cases 7 and 9), were they repeated more than once. In one case the patient apparently recovered from an attack of blood-poisoning secondary to the amputation, and died from pyæmia coming on sometime after from a bedsore. The veins leading from the stump were healthy, while those from the bedsore contained softening clot (Case 7).

The other symptoms noted amongst these nine cases were: Jaundice in one case, in which there were abscesses in the liver; diarrhœa three times; vomiting not once; sudden dyspnœa once; delirium six times; rapid emaciation four times. In the 13 cases in which post-mortem appearances are recorded, petechiæ occurred in three cases, swollen kidney in six, liver seven, spleen seven, congestion of lungs in four.

Group VI.—Cases in which, with Thrombosis and Softening of the Thrombus, Secondary Abscesses were found only in the Lungs.

(26.) Under this heading come 24* cases, of which the clinical record has been obtained in 21 cases, and the post-mortem notes in all.

* Appendix II., Table VIII. Group VI.

The primary affections in these cases were as follows:—

Amputations	9
Compound fracture	6
Joints, excision of and disease of	3
Disease of bone	2
Affections of soft parts	3
Diseases of urinary organs	1
	24

The proportion of cases of this form arising from affection of the bones contrasts strongly with that in the table of primary diseases for Group I, *a* and *b*. In this group 20 out of 24 arose from diseases affecting the bones and joints; in the first group only 23 out of 50. The time of invasion after the commencement of the primary disease could be ascertained only in 14 cases. In five it began before the end of the third day, in four more before the end of the first week, and in the rest at varying times up the 39th day.

The duration of the case after the first distinct symptoms was ascertained in 19 cases, with the following result:—

2 days	1 case.
3 „	4 cases.
5 „	2 „
6 „	3 „
7 „	1 case.
8 „	1 „
9 „	1 „
10 „	1 „
12 „	2 cases.
18 „	1 case.
19 „	1 „
32 „	1 „

On comparing this with the table for the cases of "septicæmia," it will be seen that the difference in duration is not sufficient to justify the theory that those cases which die without secondary abscesses would have developed them had they lived longer. Amongst the chief symptoms in the 21 cases of which the clinical record was obtained were the following: Rigors were wanting only in six cases, and three of these were young children; diarrhœa is noted only once; delirium five times; jaundice once; marked emaciation five times; petechiæ on skin once; profuse sweating apart from rigors only once; convulsions in one child. The temperature was such as has been so frequently recorded that no comment is needed upon it. Of the post-mortem appearances, beyond the abscesses in the lung, the following are noted:—Swollen spleen in 12; swollen kidneys 13; subserous petechiæ 3. In a large proportion of the cases the post-mortem examination revealed nothing abnormal beyond the primary affection, and the thrombosis and the "abscesses" in the lungs, or at most the swelling of the spleen and kidneys was such as would be expected from the febrile condition preceding death. The pulpy spleen of septicæmia is rarely met with in this form of pyæmia. The want of these signs of blood-poisoning suggests that in many cases it may really be absent, and that the disease may be merely a local process of thrombosis followed by decomposition and breaking down of the clot, commencing from a part of the vein implicated in a foul and unhealthy wound. The putrid emboli sms from this source lodging in the lungs would set up local processes

of inflammation and suppuration, or gangrene, which might be conceived to prove fatal without the induction of any general infective process in the body. It has been part of the object of this inquiry to attempt to obtain evidence on this point by the examination of all the organs of such cases, even when they are apparently healthy.

On the other hand, those cases which were grouped in the first two classes (Pyæmia, Group 1 *a* and 1 *b*) do in all probability depend on a general infective process, as also do many of those included in Groups II., III., IV., and V., either wholly or in part. It is interesting with regard to this question to compare the post-mortem appearances of those cases in which thrombosis with disintegration of the thrombus is known to have been present with those in which it is not. Of the latter 51 cases were collected. Amongst them in eight only was the secondary affection limited to the lungs, or not quite one in 6½ cases, and possibly in some of these eight thrombosis may have existed and not been detected. In the 47 cases in which thrombosis was known to be present, the secondary mischief was limited to the lungs in 24, or more than one half. This would point apparently to a difference in nature between the two classes of cases. There seems no reason why if they are merely modifications of one disease the occurrence of thrombosis should diminish the likelihood of secondary abscesses occurring elsewhere than in the lungs; and yet if we hold them to be the same affection we are driven to the hypothesis that because the disease affects the veins and lungs the other tissues escape. The more rational explanation would seem to be that, just as before pointed out with regard to septicæmia, in which there is one affection which is not infective, in which there is no multiplication of the poison in the blood, and another in which a true infective process is established; so it may be with pyæmia. There may be a local thrombosis with softening of the thrombus and embolism from fragments of the decomposing and disintegrated clot, setting up local visceral abscesses which cause the death of the patient, and there may also be a true general infective disease, the local manifestations of which are congestions, inflammations, and suppurations disseminated through the body both in the viscera and elsewhere. At the same time there is nothing impossible in these conditions co-existing in one patient. Experiments on animals have shown (see Koch, before quoted) that an affection of the latter kind can be induced artificially, and that when it is once started it can be communicated from animal to animal by inoculation. No experiments, so far as we know, have shown that the fatal affection caused by septic embolism artificially produced is communicable by means of the blood from one animal to another. It is possible therefore that in the human subject there may exist a specific infective process having the symptoms of the affection we know as pyæmia communicable from patient to patient; and again that in other cases the disease may consist merely of local inflammations, suppurations, and necroses arising as a result of the lodgment of septic embola, but yet may be non-specific and non-communicable and unconnected with any general infective process.

Finally, with regard to the whole of the foregoing cases both of "septicæmia" and "pyæmia," it is to be noted that with the exception of three arising from diseases of the abdominal viscera and possibly some of the urinary cases, and one case of "septic infection" arising after an excision of the knee dressed antiseptically and in which the discharges were free from odour of decomposition, the secondary disease arose in every case in connexion with decomposing discharges.

Group VII.*—Infective Periostitis. Acute Necrosis.

(27.) The cases of pyæmia secondary to acute necrosis should, from a clinical and pathological point of view, be separated from those arising from external wounds, &c. No doubt the poison, whatever it may be, is disseminated in the same way as in some of the cases classed in the previous groups, but we have no evidence that it is the same in nature. Acute necrosis commences with all the characteristic signs of a fever, thus suggesting the idea of something specific in its nature. That the exudation beneath the periosteum possesses the most intensely infective properties before it can have been contaminated by the entrance of air from without, is shown by the frequency with which metastatic inflammations and suppurations, usually of embolic origin, occur before the subperiostical abscess has been opened. This is illustrated in cases 1 and 2. Case 4 occurred during the present inquiry. There was extensive thrombosis of the veins of the leg on admission. The tibia was surrounded by a large collection of pus. The patient died on the sixth day with well-marked pyæmic symptoms and post-mortem appearances. The blood and pus were examined during life, but no organisms were detected. Four and a half hours after death, jointed moving organisms were seen in the blood from the right auricle. Microscopic examination of the organs showed micrococci in the thrombosed vein and in various other parts of the body. The urine of the case was also analysed quantitatively and qualitatively. Cases 3 and 7 were also examined microscopically. Case 8 has been included amongst those of acute necrosis from its close clinical resemblance to case 7.

Group VIII.†—Infective Endocarditis. Ulcerative Endocarditis.

(28.) This should be kept distinct for the same reasons as acute necrosis. We know that the secondary abscesses are due in these cases to a great extent to embolism, but we have no accurate knowledge of what renders the embola infective. The fact that organisms have been found in the vegetations attached to the valves is well known, but the part they play in the process is not certain. Only two cases of this affection came under the observation of the Committee during the present inquiry, for one of which they are indebted to the kindness of Dr. Greenhow.

Group IX.—Infective Myositis.

(29.) The name of infective or malignant myositis has been given by Dr. Nicaise (Revue mensuelle de Médécine et de Chirurgie, 1877) to a peculiar form of disease consisting of acute, diffuse, suppurative inflammation of various parts of the muscular system, with grave general symptoms terminating rapidly in death. One case of this rare affection occurred during the present inquiry at St. Thomas's Hospital, the notes and the microscopical examination of which will be found in Appendix IV., Case 2.

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* Appendix II., Table IX., Group VII.
† Appendix II., Table X. Group VIII.

(30.) Under these names have been described certain rare and obscure cases in which symptoms analogous to those of septic infection and pyæmia have arisen without any discoverable local source of infection. Four such cases are briefly summarised in Appendix II., Table XI. None of them occurred during the course of the present inquiry. They are merely appended as samples of the disease in question. They are not sufficiently numerous to justify any conclusions being drawn from them.

SUMMARY.

(31.) It would seem from a careful study of the cases here collected that it is probable that the diseases commonly known clinically as pyæmia and septicæmia may be grouped as follows:

1. *Septic Intoxication.*—The effects of poisoning by the chemical products of putrefaction. A non-infective disease.
2. *Septic Infection.*—A general infective process arising from the introduction of some peculiar constituent of putrid matter into the blood stream. It is supposed by some to be due to the multiplications of living organisms in the blood; by others to be the effect of a non-organised ferment. It terminates fatally without secondary inflammations.
3. *Pyæmia* (for want of a better name).—An infective process probably of a similar nature to septic infection, but differing from it in giving rise to local inflammations and suppurations often complicated with thrombosis and embolism possibly due to the blood condition.
4. *Thrombosis with softening and decomposition of the Thrombus and Embolism.*—Causing local abscesses in the viscera wherever the septic embola lodge, but without the development of any general infective process.
5. Various combinations of one or more of the foregoing conditions in the same subject.
6. Infective Periostitis.
7. Infective Endocarditis.
8. Infective Myositis.
9. A group of obscure cases in which it is impossible to form any idea as to their nature, often called Spontaneous Septicæmia or Pyæmia.

It is not intended to assert that all these varieties are proved to exist as distinct diseases, but that such a classification as the above may serve as a basis for further clinical and pathological investigation. It is quite certain that our knowledge is not yet sufficiently advanced to justify the assertion that all these various forms are only modifications of one disease. If such an assertion can ever be proved to be true, it can only be when the exact nature of every form of the disease is perfectly understood,—a time, we venture to predict, which is still very far distant.

The apparent merging of one form of septicæmia or pyæmia into another which has led some observers to such a belief may equally be explained by the co-existence of two or even more conditions in the same case. Thus a patient suffering from the effects of septic embolism may at the same time be poisoned by absorption of the chemical products of putrefaction from the wound. On this point Billroth says, "It is "in fact scarcely possible to find a name for every combination which

" occurs between sepsis, purulent infection, diffused metastatic processes.
" thrombosis, embolism, &c. We see, for instance, sepsis without any
" trace of metastasis, sepsis with diffused metastases, sepsis with
" thrombosis and embolism. We see purulent affection with diffused
" metastases, purulent infection with diffused metastases and throm-
" bosis, with thrombosis alone, with thrombosis and embolism. We
" have thrombosis with consequent local phenomena without embolisms,
" with embolisms, with hæmorrhagic effusions, with apoplexies, &c."

PART III.

Microscopical Examination of Blood, and Chemical Examination of the Urine made during Life.

(32.) The examination of the blood in cases of pyæmia and septicæmia had for its main object the determination of the presence or absence of micrococci and bacteria. At the same time any other salient features were noted, and the relative proportion of the red and white corpuscles estimated in a series of cases. It had been observed in many instances, that a very large increase of leucocytes occurred both proportionally to the red corpuscles and to the total mass of the blood; and it was desired to ascertain whether this was a constant feature, or whether the number had any special relation of time to the rigor, and also in what way this change might be related to the local symptoms. The enumeration of the red corpuscles was not attempted in any continuous series of cases, for it was found that the length of time required in any given case for a thorough examination for bacteria precluded any consecutive observations on other points. Each examination of the blood usually occupied from one to one and a half hours, or even longer, where any considerable number of observations were made.

(33.) *Method of Examination.*—In order to ensure as complete uniformity as possible the method was in nearly all cases the same. The examination was made with a Hartnack No. 10 immersion objective, a low micrometer eye-piece and a graduated draw-tube. In many cases other powers were employed, especially in the examination of bacteria, but all the results here recorded may be understood to be from examinations made in this way unless other powers are mentioned. The warm stage was used in some cases, but the practical difficulty of carrying it about constantly, and having it always ready for use where required, led to its disuse in the majority, in order to secure more uniform results. The blood examined was obtained either from the finger or the lobe of the ear. After washing the part and cleansing with absolute alcohol, it was punctured with a common sewing needle, or a broad-pointed double-edged needle; a drop of blood taken on a large coverslip and the latter dropped on to a slide, which was immediately examined. It was found that with some practice great uniformity could be attained in the thickness of the layer of blood thus examined, a single layer of corpuscles being found in every field. Every precaution was taken to ensure cleanliness and also uniformity in the mode of examination. The enumeration of the red and white corpuscles was effected by means of the *oculaire carrée* of M. Malassez. The draw-tube of the microscope having been graduated and the same lens, &c. used, it was found after a long series of observations on blood from persons in health and those suffering from various diseases, that the number of red corpuscles in a given square of similarly prepared slides was almost uniform, and that only a small difference was found if they were counted

individually or by squares, each of which contained an average of 16. It then became comparatively easy to count in a number of fields the red and white corpuscles, and to arrive at a pretty accurate estimate of their relative proportion. Everyone who has engaged in similar observations knows the many difficulties resulting from aggregations of leucocytes, currents in the fluid, and the like, but the great uniformity in the results in a given case on repeated examinations give the observations a definite value.

(34.) In the record of these observations* the precise features of the blood are noted, whether they deviate from the normal or no. This course has been followed in order that there may be no misconception of the meaning of the terms employed. The mere statement that bacteria or micrococci have been found in the blood is always open to a doubt as to what are the grounds on which the bodies in question have been so called. Hence, although it involves much repetition of normal characters, it has been thought desirable to give the notes in detail in the Appendix.

It should also be stated that in order to check the observations on the blood in pyæmia, a large number of similarly conducted observations were made upon the blood of patients suffering from other fevers and from long-continued suppuration. Scarlet fever, enteric fever, acute rheumatism, and other cases with high temperature were specially selected. These observations have not been given in detail, as they did not reveal any new facts.

(35.) The blood was examined in 18 cases. Its general character varied considerably. In some few cases it resembled anæmic blood, very scanty proportion of red corpuscles and consequent slow formation of rouleaux; in the rest, only a less degree of these characters was noted. In none of the cases were there any strikingly abnormal characters of the red blood corpuscles, such as have been stated to occur in septic diseases. The red corpuscles were not abnormally crenated and they usually formed natural rouleaux, and when measured they did not diverge from the normal size. Microcytes were rarely seen. The proportional richness in red blood corpuscles was not estimated in any series of cases, owing to the length of time necessary for the other observations on the presence or the absence of bacteria, and on the proportion of white corpuscles.

The white corpuscles were in nearly all the cases (14 out of 16) greatly in excess, both absolutely and relatively to the red. In most cases, this increase in number was not attended with any marked change in their characters. The usual variations in size were observed, but there was no sign of fatty degeneration, or of abnormal nucleation such as is seen in leucocythæmia. The amæboid movements were natural and active, in some cases unusually so.

(36.) *Proportional increase of White Corpuscles.*—The estimation of the number of white corpuscles was made by actual counting of white and red in a number of fields, and taking the average. The constancy of the results in repeated observations is a guarantee of the approximate accuracy of this method. Of the 16 cases thus examined, in only 10 were the observations sufficiently extensive and exact to allow of a precise estimate being given. Of the remaining six cases in which circumstances forbade such exact estimation:—in two there was no apparent increase; in one they appeared to be equal in number to the

* Appendix III.

white (it is not intended to assert that they were so, or even nearly so, as the mere appearance is highly fallacious); in two they were very greatly increased; and in one other case, examined shortly before death, there was enormous increase. In the 10 cases exactly estimated the proportional increase was very various. Taking the average of the total number of estimations in each case on different days, it is found that the lowest proportion of white to red observed was 1 to 100, the highest 5·6 to 100, that is from 1 white to 100 red, to 1 white to 18 red. But in eight out of the 10 cases no lower proportion than 1 to 50 was at any time observed; in five of them it was 1 to 25 or higher.

Variations in the proportions were observed in the same case at different periods, often at short intervals. Thus in one case, observed in seven successive days, the proportions were—

| | | |

a number of cases, they could not be found in all the cases, nor were they discovered constantly in those cases where at one or other time they were present. Moreover, in the same case several forms might be seen together, and the form varied considerably in different cases. It may be gathered from this that one examination of the blood could not be sufficient to exclude the possibility of the presence of bacteria, for it was found that at one time a large number, presenting certain very definite characters, were to be seen, and a few hours later the most careful search failed to detect any. The discovery of bacteria of any form in the blood is of so much importance in the light of recent investigations on putrid infection, that before discussing any of the points here raised, it will be well to summarise the results of the observations in these cases, mentioning briefly the several forms observed. In some of the cases only one form was noticed, in others two or three. It will be a question for future consideration whether these several forms are distinct, or whether they are merely stages in the development of one or the same organism. At present they may be described according to their appearance as micrococci and bacteria.

(39.) *Micrococci in the Blood.*—Although it may be presumed, from the results of the microscopical examination of the tissues, that the micrococci were probably present in the blood during life in nearly all the cases, yet their recognition is a matter of the greatest difficulty, whether they are isolated or in masses; for, although it might be possible by the aid of reagents or by cultivation to identify them, it is practically impossible by means of the microscope to distinguish them from other granules of similar size in blood examined by the ordinary methods. It is quite possible that some of the bodies described as jointed bacteria may have been linked micrococci or micrococci chains. Zoogloea masses of micrococci distinctly recognisable were not observed. In some observations which have been made upon the blood, micrococci have been stated to exist in the white corpuscles in large quantity. Such a condition was not recognised in any of the cases examined.

(40.) *Bacteria in Blood during Life:*

1. The smallest bodies observed had the form of jointed filaments of extreme tenuity composed of 2, 3, to 8 or more minute elements which, so far as they could be resolved, were themselves of ovoid or rod shape. The jointed filaments themselves measured from ·75 μ to 3· μ *, and the length of the individual elements (estimated by division of the length of the chain by the number of such elements) about·15 to ·25 (Plate I., Fig. I., *a*.). These bodies differ greatly in their appearance from the ordinary bacterium termo, not merely in size, &c., but in the apparently far lower refractive index, so that they do not stand out with so clear a definition from the surrounding fluid, and require more attentive search even when very high powers are used. That this is not due solely to their tenuity is shown by the fact that granules of no larger size appear much more distinctly. Unfortunately, at the time when these were being specially investigated the methylanilin fluid had not been tried, and they were therefore not stained, but the subsequent examination of other bodies similar in appearance in the blood of the guinea-pig in artificial septicæmia showed that these stained very readily, and were very clearly brought out by the reagent. Do these filaments possess independent movements? Upon this point a somewhat doubtful opinion must be expressed, for, although the earlier observations convinced the observer

* The now commonly used micro-millimètre designated by the sign μ, equalling $\frac{1}{1000}$ millimètre, is here employed as the unit of measurement.

that such movements occurred, it is open to question whether the movements seen were not due to currents in the fluid. Both in blood and pus, in which they were also found, such movements continued some time after coagulation had occurred, but considering that there must have been fluid in the interstices of the fibrin filaments to allow of their movements at all, and they could readily pass through these spaces owing to their minute size, it is at least doubtful how far the movements observed were not produced by such currents. The extreme minuteness of the bodies was such as to render impossible any observation such as can be made on bacterium termo. Although these organisms were not observed in all cases, in four they were carefully watched and measured, in one* they were very minutely observed at three different periods in the blood, and that not only with one specimen, but with specimens from different parts (ear and finger), and they were also found at one time in the exudation from the granulations of the stump. It is uncertain whether these bacteria are mere micrococcus chains, or whether they consist of jointed filaments; the latter seems most probable.

2. Ovoid bodies, measuring from ·3 μ to ·8 μ, were found to be of pretty frequent occurrence. These possess no special character. In some cases two similar bodies were found joined together, and presented doubtful locomotive movements.

3. Other large spherical or ovoid granules, often seen in association with the preceding, measured 1·5 μ to 2 μ. These were rarely or never seen forming chains or linked together, but in some cases† clumps of similar granules forming zoogloea-like masses were observed in considerable number. It is noteworthy that this was the only form observed in the case of recovery in which a severe rigor had occurred, and amputation was at once performed.

4. Dumb-bell bacteria were observed in only a few cases, not by any means frequently. In a case of myositis they were seen in the blood in large quantities by Mr. Wagstaffe. The bacteria usually described as dumb-bells are, properly speaking, micrococci joined in pairs. But it is probable that other forms are occasionally spoken of as dumb-bells, either short-rods in active oscillatory movement or ordinary bacteria undergoing division. If the bodies observed in this case‡ were really micrococci, the result of the microscopic examination of the heart would be very remarkable, only rod-shaped bacteria being found there.

5. Rod-shaped bacteria (bacterium termo), Plate I., Fig. 1, *b*, were seen in the majority of cases in which any form was observed. But even where present most repeatedly and in the greatest number they were not constantly observed,—perhaps owing to want of a sufficient number of observations. These bacteria measure from 1·5 μ to 2 μ on an average, but some are 3 μ or even 4·5 μ in length, and about ·4 or ·5 in diameter. They move freely with the usual well-recognised characters swimming straight forwards with an oscillatory motion. (In many observations they were seen to have a great tendency to attach themselves endwise to the coverslip, so that only one end is for a time seen. This may perhaps be due to the electric condition induced by rubbing the coverslip with a silk handkerchief kept for the purpose.) Still bacteria were very seldom seen; had they been present they might very probably have been overlooked unless present in large quantity, as the blood was not in the majority of cases stained.

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* Appendix III., Case 4.
† Appendix III., Cases 1, 14, 17.
‡ Appendix III., Case 6.

6. In one or two cases other bodies were observed which did not correspond to any of the ordinary forms of bacteria, and may possibly be of an infusorial nature. These were protoplasmic masses measuring about 6 μ to 8 μ or rather more in length, and 3 μ to 4 μ at the widest part. The extremities taper to a point, one extremity being usually more filiform and curved on one side. They sometimes presented two or three bright dots arranged in a line in the centre. They appeared to move about freely, giving out amæboid processes, but not swimming in the same way as bacteria. These bodies were observed in three cases, one of which was a case of perinæal abscess. Only very few were seen in any of the cases. As to their nature much doubt must exist until they have been more frequently observed. They would seem to be rather of amæboid than bacterial nature.

(41.) If we now classify the eight cases in which bacteria were observed, we find that in three the form of bacteria present not mentioned. In one case only dumb-bells were seen in large number. In one case only isolated granules, corresponding with large micrococci or spores in size and appearances, were seen. In another case similar spores, isolated or in clusters, were found. In four cases not only were there bacteria, but they presented the most varied forms. In one of these,* both the globular form (micrococci) and rod-shaped (bacterium termo) were found; and on one occasion still smaller rods, but not so small as were seen in some other cases. In the rest globular forms in pairs and chains, and also both very minute and ordinary rod-shaped forms were seen often side by side. It would thus appear that so far as these observations go there is no one form of bacterium peculiar to any one kind of disease. These four cases were very distinct in their clinical and pathological features. One was a case of acute necrosis, with multiple suppuration; another of amputation, followed by osteo myelitis, with typical symptoms of pyæmia, but in which no secondary abscesses were found after death; a third a very chronic case of punctured wound, with ultimate formation of an abscess of the brain only; and the last one clinically of pyæmia following amputation of the thigh, but without secondary pyæmic abscesses.

(42.) *Examination of the Urine.*—The urine was examined in 10 cases, and in two a complete quantitative estimation of its constituents was made. No special chemical characteristic was observed in any case to distinguish the urine from that of any acute form of pyrexia. The urinary water was about normal in quantity in the majority of cases, but there were frequent variations from day to day, with a tendency however to fall below, the standard being evidently maintained by the ingestion of large quantities of beef tea, milk, &c. The reaction of the urine, when opportunity afforded for testing immediately after emission, was generally acid, but it speedily became ammoniacal. The total urinary solids were below the normal, except in those cases in which albumen, or pus or mucus derived from the urinary passages, was present to any great extent. The organic solids were rather above the normal, owing to the increase in the excretion of urea and uric ucid. In the cases observed the increase of the former was about a fourth and of the latter double the average normal excretion.† Albumen was present in all cases, except two; the quantity was never excessive. Owing to the intense alkaline reaction of the urine, which so soon develops after its emission in these cases, its presence is often masked

* Appendix III., Case 3.

† It should be stated that no rapidly fatal case came under observation.

till the urine has been neutralised by the careful addition of dilute acetic acid. Blood was present in one case, and bile pigment was also found in two.* The inorganic constituents were diminished in quantity. This was notably the case with the earthy phosphates, the phosphoric acid in combination with the earthy bases in a case of ulcerative endocarditis being 0·362 grm., compared with 1·7 grm. of phosphoric acid combined with the alkaline oxides. In a case of acute necrosis it was 0·381 grm. compared with 3·6 grms.; and in a case of pyæmia following castration 0·12 grm. as compared with 1·25 grm. Chlorine was also found in lessened quantities, the highest amount observed being 2·4 grms. It has been thought probable that, owing to the destruction or diminution of the red blood corpuscles, an increase of potash salts would be found in the urine. Two estimations in typical cases were made to ascertain this point. In both the potash followed the example of the other inorganic constituents, and was found below the normal average excretion, the amounts respectively being 1·6 grm. and 1·1 grm. In this respect there is an analogy between pyæmia and scurvy, for in both there is a diminution in the number of red blood corpuscles, and a diminished excretion of potash in the urine. In scurvy it is probable that the potash is retained in the blood to maintain the alkalinity of the blood, lessened by the withdrawal of the alkaline carbonates of the food;† whilst in pyæmia the increase in the number of white corpuscles may account for its not being present in greater quantity in the urine after the destruction of the red corpuscles. Moreover it will be of interest to note in future inquiry if the reduction of the earthy phosphates occurs constantly in this class of disease; and also to inquire if the general reduction of the inorganic constituents of the urine was due to lessened ingestion in the food and drink, or to a retention in the system of these substances owing to a change in the chemical composition of the tissues, and the furnishing the requisite material for the growth and nutrition of organisms within the body.

PART IV.

Microscopic Examination of Organs.

(43.) The microscopic examinations were conducted as follows:—Of cases alleged to have died from pyæmia portions of brain; spinal cord; thyroid gland; heart and large vessels; lungs; liver; spleen; intestine; supra renal capsule; kidney; bladder; prostate; testicle; lymphatic glands; blood clot taken from heart and veins; the tissues of the wound; and adjacent fleshy parts; periosteum and the vessels leading from the wound: also in other cases locally inflamed parts not due to operation, but apparently the source of infection; muscles in parts distant from the primary lesion; the skin and the subcutaneous tissue were placed, as soon after death as possible, in a preservative fluid composed of two parts of one sixth per cent. chromic acid and one part of methylated spirit. The fluid was changed at intervals of about three days, and after three weeks replaced by methylated spirit, in which the specimens were kept until ready for section. In all cases parts were taken which seemed to the naked eye

* Appendix II., Table X., Case 1. The second case is not included in the Tables as the patient recovered; it was one of septicæmia at the Seamen's Hospital.

† Inquiry into the General Pathology of Scurvy. Charles H. Ralfe, M.D. 1877. Lewis, London.

normal as well as parts which were evidently diseased. The sections for the most part were stained with hæmatoxylin; of these some were mounted after the treatment with alcohol and oil of cloves in a solution of Canada balsam in turpentine and chloroform, others in glycerine. Koch's * method of treating the section was employed in a few of the cases examined.

The results may be stated as follows :—

(44.) 1. *Micrococci.*—In a large number of cases these organisms were found in all or some of the following parts: thyroid gland, heart, lung, liver, kidney, supra renal capsule, spleen, lymphatic glands, skin, muscles, and blood clot. They were nearly always in blood vessels and chiefly in capillaries. They occur as dense aggregations of very minute spherical bodies imbedded in a homogeneous substance which takes a deep violet stain with hæmatoxylin, being similar to that taken by albuminous casts in uriniferous tubules. In capillaries these masses completely plug the vessel, and frequently present irregular bulgings producing a varicose condition of the containing vessel. In some instances the vessel appears to have been ruptured or partly absorbed, so that the micrococci extend for some little distance over the contiguous tissues. It is noteworthy that while sections in which these organisms are found in many parts show great pathological changes, sometimes the immediate surroundings of the micrococci often present little if any alteration from the normal condition. In large vessels the micrococci either form a layer of uniform thickness adherent to the wall of the vessel and often covered with coagulated blood, or present a somewhat papilliform appearance, projecting into the lumen of the vessel with a rounded extremity surrounded with blood corpuscles and connected with the wall of the vessel by a narrow pedicle. Occasionally they are met with quite detached from the wall of the vessel and completely surrounded with coagulated blood. Their distribution is most unequal. In some cases every section of the kidney contains one or more groups of micrococci, while none can be discovered in the heart. In other cases many can be found in the heart, while they are so sparsely present in the kidney as only to be discovered in very small quantity after repeated and careful examination. Similar variations may be observed in the viscera. It is of course possible that some other parts of a viscus than that taken for examination may have contained micrococci. It seems, however, more probable that some precedent change in the chemical composition of the tissues may be necessary for the growth and multiplication of these organisms, and that thus they may be evidences rather than causes of pyæmia. Germs having got into the system—in traumatic cases by the wound, in non-traumatic cases in some other way,—might circulate with the blood, and perish from inanition unless they found, as the result of some chemical change in the tissues, a suitable index for propagation. This suggestion would harmonize with their unequal distribution, and their absence from more extensively diseased parts.

(45.) 2. *Bacteria* were found in three cases. In one,† in the glomerulus of a kidney, a vessel was distended to about three times its

* This consists in staining the section deeply with methylaniline violet, soaking in dilute acetic acid, washing in solution of carbonate of soda, and subsequently mounting by means of alcohol, oil of cloves and Canada balsam in the usual way. Equally good results were obtained by soaking in acetic acid, washing thoroughly in distilled water, without the use of alkali.

† Vide Appendix IV., Case 28.

normal size and filled with rod-like bodies, which also protruded through a rupture in the vessel and formed an irregular mass in the space between the glomerulus and the Malpighian capsule. In this case no other bacteria were found in sections taken from other parts of the kidney or of the other viscera, although these showed considerable pathological changes. It must be stated, however, that the epithelium of the kidney was much decomposed so that these bacteria may have been of post-mortem origin. In the second case, rod-shaped bacteria were found in the heart of a patient who died of acute suppurative myositis.* The naked-eye appearances showed only one or two minute subpericardial ecchymoses, but no pericarditis. The muscular tissue of the wall of the left ventricle at its upper part was of a pale yellowish white colour, with scattered ecchymoses and a somewhat mottled appearance: the surface of the section somewhat granular in parts, but no suppuration was visible, nor endocarditis. The appearance was similar to that seen in acute myocarditis. On microscopic examination of the affected parts there were found to be areas extensively infiltrated with bacteria, especially around the vessels. In some (Plate I., Figs. 2 and 3) of the smaller arterioles colonies or zoogloea masses were found, whilst the perivascular spaces around some of the larger arterioles were crowded with bacteria. They extended also in some places between the muscular bundles, but hardly any were seen within the sarcolemma. The muscles in the most infiltrated parts were degenerated in such a way as to become very deeply stained with methylviolet, the striæ less distinct, and the muscle appeared as if undergoing a sort of liquefaction; but there was no sign of granular disintegration. This condition of the muscle was seen only in some parts, especially where the bacterial infiltration was most advanced, and in some other parts where fewer bacteria were present the muscular fibre appeared quite healthy. The remainder of the muscular tissue appeared absolutely healthy. The bacteria differed in no important respect from ordinary bacterium termo. The majority of them were rods of length varying from 1·5 to 3 μ; some isolated, other joined in pairs end to end; a few formed more elongated filaments with or without traces of division, and in some of these spores were visible with high powers. Micrococci were also seen in this heart, one or two zoogloea masses only being found. In this case it might likewise be suggested that these bacteria were merely the result of post-mortem decomposition. The following reasons are, however, against such a supposition, for they were not found in any of the other organs similarly examined, even in the muscular abscesses in the arm; nor was there evidence of decomposition of the heart either as seen with the naked eye or with the microscope, and in the organs which appeared more decomposed bacteria were not found. The change may therefore be regarded as occurring during life, but whether under the influence of the bacteria must be doubtful.† Notes of the third case in which bacteria were found are recorded in Appendix IV., Case 30.

(46.) 3. *Changes in Blood Vessels.*—In almost all the tissues the endothelium of the blood vessels was found detached to a greater or

* Vide Appendix IV., Case 2.

† Rindfleisch has described a similar change in the heart, which he says is met with in pyæmic, puerperal, and glandrous infections. The "vibrios" are at first crowded together between the muscular bundles and later penetrate into their interior, and the muscular fibres become dissolved. He states also that they never lead to actual suppuration, owing to the rapidly fatal issue of the diseases in which they occur.

less extent from the walls of the vessels. In some cases this detachment had taken place *en masse*, so that the collapsed tube of endothelium lay free on the lumen of the vessel. In other cases the separate endothelial cells were found intermixed with blood corpuscles. Frequently the deeper layers of the intima were much hypertrophied, and this change involved in some cases the entire circumference of the vessel equally, in other case irregularly. This hypertrophy produced corresponding diminution of the lumen of the vessels, in varying degrees even to complete obliteration (Plate III., Figs. 2 and 3). Again, this hypertrophy was sometimes followed by degeneration, so that the intima appeared as a thick homogeneous layer which stained violet with hæmatoxylin. This change was especially noticeable in the glomeruli of the kidney, in the description of which organ special reference will be made further on. In other cases the external coat of the vessels was hypertrophied, and this again was frequently degenerated like the intima, the fibres being swollen and homogeneous in appearance, and staining similarly with hæmatoxylin. The hypertrophy sometimes encroached on the other parts of the vessel, sometimes on the surrounding tissues, or on both, so that masses of degenerative fibrous tissue would remain with either no indication of vascular origin, or only a narrow cleft lined with endothelium (Plate VI., Fig. 2), to mark the tract of the vessel. In the venules no change like obliterative endo-arteritis was noticed. Thrombi were very common. Inflammatory processes resulting from ulceration of the vein walls were also seen in some parts. The veins which contained softening thrombi did not give at all satisfactory results, owing to the softness and ready separation of the contents during the process of preparation. A certain number of sections of the femoral and of the portal veins in cases of suppurative phlebitis were, however, examined both by Koch's method and with logwood; but nothing special was observed. The walls of the vein were much thickened, the endothelium completely detached, and often also the inner coat was entirely destroyed, the vessel containing only a mass of altered blood, in which in some cases micrococci were found.

No special change in the walls of the veins was observed except such as were associated with inflammation. Thrombosis of minute venules, not connected with any visible change in their walls, or around them, was observed in a large number of organs; it was found in the brain, lungs, heart, liver, kidney, and skin. The thrombi were mainly comprised of leucocytes with fibrin; sometimes they occupied the entire channel, occasionally only a part leaving a passage on one side. These thrombi as a rule appeared to be recent, but in some parts they seemed to be undergoing organisation. Connected with these thrombi, minute abscesses which were found in various organs, and appeared dependent on the thrombosis. They varied in stage and size from slight exudation around the vessels as seen in the brain, to pin-point abcesses in several organs, and to small pustules in the skin. In some of these, when in an early stage, the wall of the vein could still be distinguished in the centre of the abscess; in others it was completely destroyed, but the position of the vessels was indicated by the anatomical situation (Plate IV., Fig. 1), the corresponding artery lying alongside the abscess, its walls having undergone obliterative changes.

(47.) 4. *Connective Tissue.*—This in all organs had undergone changes; in some parts merely an increase of nuclei, in other considerable hypertrophy, and in others miliary abscesses.

In some places, especially in the fibrous layer of the corium, the periosteum, and the fibrous coat of vessels, there appeared a swollen condition of the fibrous bundles, limited to comparatively small tracts; the affected parts stained very deeply with reagents, and the connective tissue corpuscles lying between these bundles also stained very deeply and had a more granular appearance.

(48.) 5. *General changes* may be briefly summarised as consisting of granular and fatty degeneration of epithelium, excessive pigmentation of epithelium and nuclei, and congestion of blood vessels with extravasations of blood.

(49.) *Liver.*—In this organ micrococci were frequently found, but not so frequently as in the kidney. They were mostly in capillaries, but sometimes in larger vessels. In addition to changes such as have been before described in the vessels, and in the connective tissue, it is to be observed that frequently a single acinus will have undergone excessive degeneration, while the surrounding acini present little if any change. Thus in a section an acinus would be seen with the hepatic cells shrivelled and granular, and the sustentacular structure swollen, while the surrounding acini presented no other change than perhaps some swelling of the hepatic cells, and an increased quantity of pigment. Such a condition is probably of embolic origin. Again hepatic cells are often to be found with their substance almost entirely replaced by fat, and this change may either have involved all the cells in different acini. In the latter case the section, after treatment with alcohol and oil of cloves, appears as if riddled with holes, which on examination prove to be hepatic cells with their protoplasm forced to the peripheries, and the fat which had filled the cell dissolved out. Sometimes plumose crystals of tyrosin were seen in the veins.

In only three out of 30 livers examined were abscesses visible with the naked eye; a part of the wall of one of these abscesses contained micrococcus clumps, and these were cultivated in aqueous humour at a temperature of 36° C. and produced in two generations only micrococci. Nothing else of a special nature was seen in these abscesses. In all the livers examined some exudation in the portal spaces was discovered; in one case of acute septicæmia it reached an extreme degree. In this case there was also a very remarkable condition of the hepatic cells, their protoplasm appeared to have undergone a sort of fragmentation, and had a glassy look, but the cells had not become broken down (Plate V., Figs. 1–4). In the other cases there was only some slight swelling of the liver cells here and there. Fatty degeneration seen in some cases was judged to be due to antecedent conditions from the state of other organs. Thrombi were found in small vessels here and there, just as is noticed in the kidney. In some cases the intralobular vein was also compressed by thickened connective tissue.

(50.) *Kidney.*—This organ was more generally affected than any other. Micrococci are almost always found, but, as before stated, in very unequal quantities. The capillaries surrounding the straight tubules in the upper part of the Malpighian pyramids appear to be a favourite position for them; but they are also to be met with in those of the cortical substance, and sometimes in some of the vessels of glomerulus. In addition to the changes in blood vessels before described, the glomeruli are often greatly altered. In some an increase of nuclei is chiefly noticeable. In some cases this reached such a degree as to obscure the capilsaries, and in two cases there was also extensive exudation in the surrounding tissues, similar to that seen in scarlatina nephritis. In

some also the walls of the component vessels were much thickened and stained deeply with hæmatoxylin. In others this thickening has resulted in complete obliteration of the lumen of the vessels, and this change may either be limited to a part of the glomerulus, or involve it uniformly. A glomerulus thus changed appears to be welded into a homogeneous mass, which in later stages of degeneration is much shrunken, and no longer colours with hæmatoxylin. Laminated fibrine is sometimes found between the glomerulus and the Malpighian capsule. A change was often observed in the afferent arterioles; their walls appeared to be infiltrated, whether with leucocytes or by proliferation of their own cells could not always be determined; but in some cases the endothelium was swollen, and projected into the lumen so as nearly to close it. In one case the space between the glomerulus and capsule was in many Malpighian bodies filled up with granular exudation, which did not appear to consist of or to contain micrococci. Changes in the capsule and its endothelium were rarely observed; where they existed there was evidence that they were chronic, and therefore independent of the disease under consideration. A marked change seen in all the cases consisted in swelling of the epithelium of the convoluted tubes, with various degrees of degeneration. In some this was only moderate, the outline of the swollen epithelial cells being distinctly visible; in others the tubes were filled with degenerated epithelium or a granular mass, which in one or two was of fibrinous appearance. The most extreme degree of change was seen in a case of acute septicæmia; in this the epithelium was swollen so as completely to close the tubule, the cells appeared to have undergone a fragmentation of their protoplasm, especially where it was in contact with the basement membrane (Plate IV., Fig. 2), whilst the more superficial parts were fused together into a granular mass, which occupied the centre of the tubules.

Thrombi were occasionally found in the veins, especially in the *venulæ rectæ*, near the base of the pyramids. In some cases the basement membrane of the uriniferous tubules has undergone a change similar to that described in the intima of the blood vessels. In the collecting tubes the basement membrane sends in processes between the epithelial cells, which are most distinctly seen where the loosened epithelium has fallen out (Plate IX., Fig. 2). When this has occurred the wall of the tube seen in profile presents a crenated margin towards the lumen of the tube, but when the section has exposed the surface of a tube, these processes appear as lines passing completely across the tube, like rungs of a ladder. This arrangement, so far as can be ascertained, has not been hitherto described, but since attention was accidentally directed to it in the course of the inquiry, it has been observed more or less distinctly in all the specimens examined. Normally these processes are colourless, like the membrane of which they are a part, but occasionally they are to be found thickened, and of a deep violet colour (Plate IX., Figs. 3 and 4). Some of the collecting tubes are often plugged with masses composed of small spherical bodies, colourless in the centre, and stained violet at the periphery. These dissolve without effervescence on the addition of dilute hydrochloric acid. The remaining changes observed in pyæmic kidneys may be summed up as follows:—1. Considerable engorgement of superficial blood vessels. 2. Extravasated blood into and between uriniferous tubules. 3. Fibrinous plugs in tubules. 4. Granular deposit (?) of yellow pigment in epithelium.

(51.) *Heart and Pericardium.*—In two cases of ulcerative endocarditis micrococci were found in the substance of the valves, and in the vegetations adherent thereto. In one case of pyæmia of traumatic origin the heart was found with numerous colonies of micrococci in the capillaries. The connective tissue showed greatly increased cell-growth, and in many places miliary abscesses. The muscular fibres were in some parts quite normal. In others the fibres presented transverse bands, with thick well-marked borders, suggesting an exaggerated form of the normal junction of cardiac muscle fibres. On closer examination these bands seemed to be the result of rupture of the more superficial part of the fibre. The pale band was thus formed by the unruptured portion of the fibre, and the well-defined borders by the retraction of the ruptured superficial parts (Plate VIII., Figs. 1 and 2). In other places this change was found in a later stage, the bands much broader, and the mode of origin more evident. In other parts, again, the fibres were found completely transformed into granular matter. The change in the heart in a case of myositis has been described when speaking of bacteria (§ 45). In another case of acute necrosis with commencing pericarditis, the changes observed were limited to the surface of the heart and pericardium. The muscular fibres were separated by fibrinous exudation; thrombi composed of leucocytes were found in the small vessels both of the heart and the subpericardial fat. Scattered through the more superficial layer of the pericardium were seen masses of micrococci, which preserved an intense staining after treatment with methylanilin and acetic acid. Some of these were distinctly in vessels; others did not appear to be surrounded by any vessel wall. Small clumps and layers of micrococci were also scattered near the surface. (Plate II., Fig. 2. Plate III., Fig. 1.)

(52.) *Lungs.*—In the lungs vessels were often found plugged with coagulum and extravasated blood in surrounding structures. Bronchopneumonia in varying stages was also of frequent occurrence. Micrococci were found either in capillaries surrounding an alveolus or in large vessels in the subpleural tissue. Sometimes they were found in groups outside blood-vessels, and the specimens show very clearly the origin of this in Plate XI., Fig. 1. A large vein gave off a small branch, which at one part had undergone partial absorption of its wall. Through this micrococci had escaped, pushing the adventitious tunic before them, and had developed into large masses in the spaces filled with extravasated blood. The pleural surface was in many cases much thickened, and this had either occurred uniformly or else at certain points, which thus on section presented a papillary appearance. This latter was the result probably of distension of the subpleural lymphatics.

(53.) *Lymphatic Glands.*—These in some cases contained micrococci in the blood-vessels. Many of the blood-vessels showed marked hypertrophy and swelling of the adventitia. In one gland were large numbers of giant cells, and Hassell's corpuscles, such as Lang also found in glands in cases of lupus.

(54.) *Spleen, Thyroid Gland, Supra renal Capsules.*—Plugs of micrococci were found in the spleen in some of the cases (Plate I., Fig. 5). In others considerable changes had occurred in the blood-vessels similar to those noticed in other viscera, to which reference has been already made. Micrococci were also found in the thyroid gland and suprarenal capsules. In nearly all cases where they were examined there was considerably increased cell-growth in the connective tissue. In one case the thyroid was full of minute abscesses.

(55.) *Brain or Spinal Cord.* — The noticeable features were the presence in large quantities of so-called amyloid bodies, and in one case minute abscesses were found scattered through the cortical substance.

Skin.—In two cases the skin presented scattered minute abscesses and pustules, some of which were deep, others were superficial; they varied from one m. m. to three or four in diameter. The arterioles showed in a very marked degree the proliferation of the inner coat (Plate III., Fig. 3). The abscesses contain broken-down pus, with many ovoid or rounded bodies measuring two or three m. m. in diameter, (Plate IV., Fig. 1), staining deeply with reagents; and also micrococci in links and chains, but no zooglœa masses were observed.

(56.) *Muscles* (other than the heart).—The muscular tissue showed no special change in parts where no inflammatory or suppurative lesions existed. Abscesses, of sizes varying from that of a pin's head upwards, and tracts of scattered inflammation in their vicinity, were examined; in the latter exudation, mainly consisting of fibrinous material, was found, separating the muscular bundles; leucocytes and fine granules were also present in varying quantities. In other parts the muscular fibres were separated by masses of leucocytes, as in ordinary suppurative inflammation. Small hæmorrhages were not infrequent (Plate II., Fig. 1). Micrococci were found in the vessels as in other parts, but only in very small number. In the exudation were often seen isolated pairs of micrococci, though no rod-shaped bacteria. Zooglæa masses were found in one case in the inflamed muscle (Plate I., Fig. 6). The muscular fibres in the affected tracts were found in various stages of degeneration, such as are usually seen in myositis; the striation being first lost, and the fibre being converted into a homogeneous mass in which clear spaces occurred, followed by a gradual breaking down of the muscular tissue. The peculiar transverse markings noticed in the muscular tissue of the heart were also seen in some places; the change appeared to be due to a rupture of the degenerated fibre within the sarcolemma.

Marcus Beck.
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Charles Henry Ralfe, Secretary.

App. B., No. 4

Report on Pyæmia, Septicæmia, and Purulent Infection.

APPENDIX I.

St. George's Hospital.—Fatal Cases of Pyæmia, 1869–78.*

Total Number of Cases.

Males	64
Females	21
	85

	1869	1870	1871	1872	1873	1874†	1875	1876	1877	1878
Males	7	14	7	6	10	5	6	3	3	3
Females	4	1	4	1	1	3	2	2	2	1
Total	11	15	11	7	11	8	8	5	5	4

Year.	Jan.		Feb.		Mar.		April.		May.		June.		July.		Aug.		Sept.		Oct.		Nov.		Dec.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1869	2	–	2	–	–	–	1	–	–	–	–	–	–	–	–	1	–	1	–	1	1	1	1	–
1870	1	–	3	1	1	–	–	–	2	–	1	–	–	–	2	–	1	–	–	–	2	–	1	–
1871	–	–	1	1	1	–	–	–	2	–	1	–	1	2	–	–	–	–	–	–	–	1	–	–
1872	1	–	–	–	1	–	–	–	1	–	1	–	2	–	–	–	–	1	–	–	–	–	–	–
1873	–	1	2	–	1	–	2	–	1	–	–	–	2	–	–	–	–	–	–	–	1	–	1	–
1874	–	2	–	–	1	–	–	–	2	–	†	†	†	†	†	†	–	1	1	–	–	–	1	–
1875	–	–	–	–	1	–	1	–	–	–	–	–	1	–	1	1	–	–	–	–	2	–	–	1
1876	1	–	–	–	–	–	–	–	–	1	–	1	2	–	–	–	–	–	–	–	–	–	–	–
1877	–	–	2	–	–	1	–	–	–	1	–	–	–	–	1	–	–	–	–	–	–	–	–	–
1878	–	–	–	–	–	–	–	1	–	–	1	–	2	–	–	–	–	–	–	–	–	–	–	–
Total	8		12		7		5		10		5		12		6		4		2		8		5	

* Compiled from the post-mortem book, St. George's Hospital. (C. H. R.)

† Hospital closed for repairs and alteration of system of drainage (during June, July, and August).

Details of the 85 Cases.

——	Compound Fractures or Dislocations, Lower Extremity.	Compound Fractures or Dislocations, Upper Extremity.	Amputations of Lower Limb.	Amputations of Upper Limb.	Simple Fractures of Lower Limb.	Simple Fractures of Upper Limb.
Male	9	—	8	2	—	1
Female	1	—	1	—	—	—
Total	10	—	9	2	—	1

——	Joint Disease of Lower Limb.	Lacerated Wounds of Upper Extremity.	Abscess of Upper Segment.	Abscess of Lower Segment.	Caries of Vertebræ, or Psoas Abscess.	Anthrax.
Male	3	1	3	3	3	—
Female	2	—	1	—	—	1
Total	5	1	4	3	3	1

——	Bone Diseases, Lower Limb.	Bone Diseases, Upper Limb.	Lacerated Scalp.	Fractured Skull with or without Trephining.	Fractured Ribs.	Osteomyelitis.
Male	2	1	1	3	1	1
Female	3	—	1	2	—	1
Total	5	1	2	5	1	1

——	Amputation of Breast.	Lithotrity.	Ulcers.	Ligature of Arteries.	Removal of Simple Tumour.	Urethral.
Male	—	1	1	1	1	7
Female	1	—	1	—	1	—
Total	1	1	2	1	2	7

——	Piles, Removal.	Excision of Joint.	Various.	No Cause.	Herniotomy.
Male	2	1	3	9	1
Female	1	—	—	—	—
Total	3	1	3	9	1

NUMBER OF PATIENTS AT EACH AGE.

	Ages in Years.									
	1–10.	10–20.	20–30.	30–40.	40–50.	50–60.	60–70.	70–80.	80–90.	Not stated.	Totals.
Males - -	1	7	10	12	18	10	3	–	–	3	64
Females - -	1	2	6	4	3	3	1	–	1	–	21
Total - -	2	9	16	16	21	13	4	–	1	3	85

In the 85 cases pyæmia followed—

Operation in - - - - -	21 cases.
Injury „ - - - - -	20 „
Urethral „ - - - - -	7 „
Disease „ - - - - -	28 „
Uncertain „ - - - - -	9 „

In 42 cases the average stay in hospital of patient before development of disease was - - - - 12 days.

In 48 cases the average duration of the disease from invasion to death was - - - 12·1 „

RELATION BETWEEN THE CASES OF ERYSIPELAS AND THE CASES OF PYÆMIA.

	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878
Pyæmia - -	11	15	11	7	11	8*	8	5	5	4
Erysipelas† - -	35	20	38	18	23	61*	42	60	60	–

TABLES OF DEATHS FROM PYÆMIA AND SEPTICÆMIA ENTERED AS SUCH IN THE DEATH-BOOK OF GUY'S HOSPITAL IN THE YEARS 1869–1879.‡

	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	Total.
Primary amputation and compound fracture.	1	6	4	3	2	1	2	1	2	2	24
Secondary amputation for disease.	2	2	3	2	4	3	5	2	4	2	29
Urinary diseases -	2	5	3	4	5	4	5	4	–	2	34
Diseases of bone, including fractures (simple).	2	1	5	3	5	9	2	4	9	1	41
Diseases of joints, including excisions.	1	2	2	1	5	3	4	2	–	2	23

* Hospital closed for three months from June.

† Cutaneous erysipelas, phlegmonous erysipelas, diffuse cellulitis.

‡ Compiled by Mr. G. A. Wright, Surgical Registrar.

——	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	Total.
Osteotomy	–	–	–	–	–	–	–	1	–	–	1
Carbuncle	1	–	1	2	–	–	2	1	–	–	7
Hernia operations	1	–	–	–	–	–	–	–	–	–	1
Ulcers	1	–	1	–	–	–	–	–	–	–	2
Gangrene	3	–	–	–	–	–	1	–	–	1	5
No cause	2	1	1	–	–	–	–	–	–	–	4
Cellulitis	2	1	–	–	–	–	1	1	–	–	5
*Erysipelas	–	–	–	–	2	–	3	1	1	1	8
*Puerperal and uterine	2	2	1	2	1	2	1	1	3	–	15
Wounds	1	1	4	3	2	2	4	2	3	–	22
Ulcer of rectum	–	–	–	–	–	–	1	1	–	–	2
,, pharynx	1	–	–	–	–	–	–	–	–	–	1
,, colon	–	1	–	–	–	–	–	–	–	–	1
,, duodenum	–	–	–	–	–	–	1	–	–	–	1
*Scarlatinal dropsy	–	1	–	1	–	–	–	–	–	–	2
Idiopathic	1	2	–	1	2	–	4	1	–	–	11
Operation for epithelioma of face, &c.	–	–	–	–	1	3	–	–	–	–	4
Operation for cancer of breast and other parts.	–	–	1	–	–	1	1	2	–	1	6
Operation for nasal polypus.	–	–	–	–	–	–	–	1	–	–	1
Suppurating eye-ball	–	–	–	–	–	1	–	–	–	–	1
Abscesses in various parts.	–	–	1	–	–	1	–	2	1	–	5
*Heart disease	–	–	1	–	–	–	1	–	–	–	2
*Typhoid	–	–	–	–	–	–	–	–	–	1	1
Exophthalmic gôitre	–	–	–	–	–	–	–	–	1	–	1
Ligature of arteries for aneurism.	–	–	1	–	–	–	–	–	1	–	2
*Burn	–	–	1	–	1	–	–	–	–	–	2
Thrombosis of portal vein.	–	–	–	–	–	–	1	–	–	–	1
Excision of nævus	–	–	–	–	–	–	–	–	1	–	1
*Paraplegia and renal disease.	–	–	–	–	–	–	2	–	–	–	2
Total	23	25	30	22	30	32	39	27	26	13	267

* These cases are not included in Tables II. and V. of Report.

App. B., No. 4.

Report on Pyæmia, Septicæmia, and Purulent Infection.

London Hospital.—Fatal Cases of Pyæmia, 1869–1878.

Total Number of Cases.

Males	149
Females	31
	180

Total Number in each Year.

	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878
Males	7	7	13	18	14	28	23	12	18	9
Females	3	—	4	4	5	1	4	4	3	3
Total	10	7	17	22	19	29	27	16	21	12

Year.	Jan.		Feb.		Mar.		April.		May.		June.		July.		Aug.		Sept.		Oct.		Nov.		Dec.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1869	1	–	1	–	–	–	–	–	–	1	3	1	1	–	–	–	–	1	–	–	1	–	–	–
1870	–	–	4	–	–	–	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
1871	3	–	3	2	–	1	1	–	–	–	1	–	3	1	–	–	–	–	–	–	2	–	–	–
1872	–	–	–	1	3	–	1	–	3	–	1	1	1	1	3	2	–	–	2	–	1	1	1	–
1873	3	–	1	–	3	2	1	1	4	1	–	–	–	–	–	–	–	1	1	–	1	–	–	–
1874	–	–	6	–	3	–	1	–	3	–	1	1	3	–	1	–	3	–	2	–	2	–	3	–
1875	–	–	4	1	3	–	–	–	2	–	4	1	–	–	–	1	3	–	3	–	2	1	2	–
1876	2	2	2	1	1	–	–	1	2	–	–	–	–	–	1	–	1	–	1	–	2	–	–	–
1877	1	–	1	–	4	–	2	–	1	–	2	–	1	–	–	–	2	1	3	–	1	1	–	1
1878	–	–	1	–	1	–	–	–	1	2	–	–	1	1	–	–	2	–	1	–	1	–	–	1
Total	12		28		21		11		20		16		13		8		14		13		16		8	

Details of the 180 Cases.

	Compound Fractures or Dislocations of Lower Limb.	Compound Fractures or Dislocations of Upper Limb.	Amputations of Lower Limb.	Amputations of Upper Limb.	Simple Fractures of Lower Limb.	Simple Fractures of Upper Limb.	Erysipelas, no Injury mentioned.
Male	16	7	23	8	5	1	7
Female	—	—	6	1	—	—	—
Total	16	7	29	9	5	1	7

——	Joint Disease, mostly suppurating, Lower Limb.	Joint Disease, mostly suppurating, Upper Limb.	Wounds, mostly lacerated, Lower Limb.	Wounds, mostly lacerated, Upper Limb.	Abscess, Upper Segment of Body.	Abscess, Lower Segment of Body.	Psoas Abscess.
Male - -	10	3	7	5	—	6	1
Female -	3	1	2	1	2	1	1
Total -	13	4	9	6	2	7	2

——	Bone Disease, Lower Limb.	Bone Disease, Upper Limb.	Lacerated Scalp.	Trephining.	Fractured Ribs.	Cancer.	Amputation of Breast.
Male - -	9	4	3	1	2	2	—
Female -	3	1	—	—	—	1	3
Total -	12	5	3	1	2	3	3

——	Anthrax.	Wounds, &c. of Perineum.	Lithotomy.	Ulcers.	Scald.	Gangrene.	Rupture of Urethra.
Male - -	3	3	2	2	1	1	1
Female -	—	2	—	—	—	1	—
Total -	3	5	2	2	1	2	1

——	Ulcer of Rectum.	Ligature of Piles.	Various.	No Cause stated.
Male - - -	1	2	3	10
Female - - -	1	1	—	—
Total - -	2	3	3	10

Number of Patients at each Age.

——	Ages in Years.														
	1–4.	5–9.	10–15.	16–20.	21–25.	26–30.	31–35.	36–40.	41–45.	46–50.	51–55.	56–60.	61–65.	66–70.	71–75.	76–80.
Male -	2	8	13	13	10	14	27	15	11	13	7	10	2	1	3	1
Female	—	5	2	2	1	6	3	4	2	1	1	3	—	1	—	1
Total	2	13	15	15	11	20	30	19	13	14	8	13	2	2	3	

The youngest patients, 4 months; 1½ years. The oldest, 76 years.

Post-mortem Notes.

No. and Date.		Sex and Age.	Nature of Case.	Notes of Autopsy
2353	1876	M. 27	Wound of thigh -	Osteo-myelitis; clot in femoral vein; pneumonia; hæmorrhages in spleen and liver.
68	„	M. 41	Wound of knee -	Pyæmic pneumonia; pus in bronchi.
2354	„	M. 60	Cellulitis of leg -	Pus in pericardium; abscesses in heart.
219	„	M. 1½	Scald, abscess in axilla	Pyæmic pleurisy and pneumonia.
743	„	M. 57	Gangrene, erysipelas -	Pleurisy; spleen very soft.
713	„	M. 23	Laceration of foot -	Abscesses in heart, lungs, liver, and kidney; pleurisy.
1189	„	M. 55	Amputation through knee.	Pneumonia; abscess in lungs; secondary abscesses in arm; nephritis.
1853	„	M. 11	Compound fracture of leg.	Osteo-myelitis; pneumonia.
2079	„	M. 45	Erysipelas of leg -	Thrombosis; pyæmia.
1021	„	F. 15	Acute periostitis of tibia	Pus in knee-joint; embolisms in lungs; pus in shoulder-joint; spleen large and soft; liver very soft.
8	„	F. 38	Amputation of right leg	Abscess in kidneys; infarcts in spleen and base of one lung; phlebitis.
215	„	F. 43	Cancer of breast, amputation.	Pus in joints and lungs.
19	1870	M. 22	Amputation of leg -	Phlebitis; pleuritic effusion; abscesses in lungs; clot (adherent) in branch of pulmonary artery.
22	„	M. 27	Fracture of leg - -	Pus in ankle-joint and joints of finger; pleurisy; conjested lungs.
26	„	M. 11	Periostitis of tibia -	Pus in knee, ankle, and elbow; pericarditis; pleurisy; infarcts in kidney.
39	„	M. 50	Carbuncle of back -	Infarcts in heart and base of lungs; congestion of kidneys.
74	„	M. 23	Suppuration of knee-joint.	Abscesses in lungs and in joints of fingers and hand; other viscera free.
79	„	M. 62	Perineal section -	Abscess in both lungs and one kidney; pleurisy; liver normal.
6	1871	M. 31	Lacerated thigh -	Abscesses in lungs; pleurisy; pericarditis; emboli in kidney and liver; spleen healthy.
21	„	M. 27	Compound fracture of leg.	Infarcts on posterior aspect of heart; abscesses in liver.
35	„	F. 16	Disorganisation of shoulder-joint.	Extravasation on pericardium; abscesses in lungs, spleen, and kidneys; disintegrated clot in iliac and femoral veins; pus in elbow-joint.

No. and Date.	Sex and Age.	Nature of Case.	Notes of Autopsy.
44 1871	M. 19	Amputation of arm -	Abscesses in lungs; extravasations on anterior and posterior surfaces of heart.
45 „	M. 66	Lithotomy - -	Pus in knee-joints, &c.
73 „	M. 12	Disease of shoulder-joint	Pericarditis; pleurisy.
107 „	F. 58	Abscess of hand -	Lungs congested; sub-pleural hæmorrhages; infarcts in spleen; abscess of brain.
115 „	M. 31	Compound fracture of humerus.	Pyæmic deposits in lungs and pericarditis.
128 „	M. 57	Ulcers of rectum -	Pericarditis; pyæmic pneumonia; abscess in front of thyroid cartilage.
167 „	F. 22	Abscess over knee -	Pleurisy; abscesses in lungs; pyæmic deposits in spleen; local peritonitis.
183 1872	M. 54	Fractured ribs - -	Pleurisy; infarcts and abscess in lungs; other viscera healthy.
192 „	M. 31	Lacerated leg - -	Pleurisy; lobular pneumonia; abscesses.
36 1870	M. 32	Amputation of leg -	Ditto.
40 „	M. 39	Compound fracture of leg.	Abscesses in liver and lungs; jaundice.
54 „	M. 39	Ditto - - -	Deposits in spleen, but in no other organ.
72 „	M. 10	Disease of ankle-joint -	Abscesses in heart; in intercostal muscles; pericarditis.
93 „	M. 50	Amputation for popliteal aneurism.	Lobular pneumonia; pleurisy, congested kidneys.
107 „	M. 14	Compound fracture of fore-arm.	Multiple abscesses in lungs and liver.
133 „	M. 60	Compound fracture of leg.	Ditto.
143 „	M. 43	Abscess in thigh -	Pus in hip and knee-joint; pleurisy; abscesses in liver, spleen, kidneys, and lungs.
210 „	M. 38	Amputation of fore-arm	Pleurisy; abscesses in lungs and liver.
42 1875	M. 33	Laceration of knee-joint suppuration.	Pleurisy; abscesses in lungs and liver.
45 „	M. 33	Severe laceration of hand, secondary amputation.	Multiple abscess in lungs.
75 „	M. 5	Acute disease of knee-joint.	"Suppurative meningitis; pyæmic abscesses."
155 „	M. 7	Trephining - -	Abscesses in liver.
221 „	M. 32	Old ulcer of leg, erysipelas.	Pleurisy; abscesses in lungs.
269 „	M. 18	Suppuration of knee -	Abscesses in lungs.
512 „	M. 13	Acute periostitis of femur.	Pus in sterno-clavicle and knee-joints.
525 „	M. 9	Hip disease - -	Pneumonia and pyæmic abscesses.
596 „	M. 15	Compound fracture of leg.	Pus in right shoulder and left knee.

No. and Date.		Sex and Age.	Nature of Case.	Notes of Autopsy.
718	1875	M. 25	Psoas abscess - -	Pus in hip; pyæmic pneumonia.
878	„	M. 4 mos.	Fractured femur -	Abscess in joints.
897	„	M. 21	Amputation of legs.	
1064	„	M. 17	Disease of knee -	Osteo-myelitis; abscesses in lungs.
1434	„	M. 33	Crush of hand - -	Pleurisy; abscesses in lungs and kidney.
1618	„	M. 47	Laceration of hand -	Pus in joints.
1693	„	M. 46	Amputation at shoulder-joint.	Abscess in knee-joint, &c.
1718	„	M. 71	Amputation of thigh -	Abscesses in liver.
1758	„	M. 33	Erysipelas - -	Pus in joints, pleura, and lungs.
1809	„	M. 38	Fractures of ribs and sternum.	Abscesses in lungs; pleurisy.
1992	„	M. 23	Compound fracture of leg.	Pus in elbow-joint.
2140	„	M. 30	Injury to knee - -	Osteo-myelitis; pleurisy, and abscesses in lungs.
88	„	F. 26	Suppuration of knee.	
261	„	F. 7	Amputation of thigh -	Osteo-myelitis; pus in pleura; lungs congested; pyæmic abscesses in one lung.
639	„	F. 37	Gangrene of fore-arm -	Pus in joints; soft spleen.
935	„	F. 7	Acute periostitis of tibia.	Pleurisy; pericarditis; abscess in heart, in kidney, and about uterus.
223	1874	M. 17	Amputation of arm -	Osteo-myelitis; effusion in pleura; lung partly collapsed, partly solidified (lobular pneumonia).
259	„	M. 31	Compound fracture into ankle-joint.	Pleurisy; pus in wrist ankle, and elbow-joints.
16	1869	M. 50	Disease of elbow-joint -	Pleurisy; lobular pneumonia.
36	„	M. 44	Lacerated scalp, ostitis of bone.	Thrombosis of superior longitudinal sinus; deposits in lungs and liver; gall-bladder full of clear glairy fluid.
77	„	M. 52	Ligature for piles -	Abscesses in lungs; fluid in knee-joint.
91	„	F. 35	Amputation of leg -	Pus in wrist-joint; congested lungs; deposits in spleen.
183	„	M. 35	Disease of lower jaw -	Pleuritic effusion; abscesses in lungs and kidney.

Full details of each of the above cases can be obtained by quoting the number and date, and of any of the cases in the years 1873, 1877, 1878.

Compiled by Frederick Treves,
Surgical Registrar London Hospital.

Analysis of Deaths from Pyæmia and Septicæmia occurring in Middlesex Hospital from 1869–1878.*

Cases admitted	Males	8
	Females	6
Cases originating	Males	34
	Females	10
Total		58

Table showing the relation between Cases originating in Hospital and those admitted Pyæmic, the Number of Operations, and Cases of Erysipelas.

—	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	Total.
Cases originating	4	5	10	5	7	5	–†	2	‡	‡	
Cases admitted	–	1	–	2	1	4	3	3	–	–	14
Number of operations	184	187	214	216	27‡	247	287	259	‡	‡	‡
Erysipelas admitted	10	13	11	38	50	46	31	18	‡	‡	‡
Erysipelas originating	12	6	13	11	19	31	23	7	‡	‡	‡

Ages of the 58 Cases.

Sex.	1–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	Total.
Males	–	4	5	14	8	8	1	2	44
Females	1	–	3	1	4	2	5	–	16
Total	1	4	8	15	12	10	6	2	58

* From Annual Report of Registrars.—C. H. R.
† Only one case occurred, which recovered.
‡ Detailed report for these years not yet published.

App. B., No. 4.

Report on Pyæmia, Septicæmia, and Purulent Infection.

Details of the 44 Cases occurring in Hospital.

—	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	Total.	
Amputation of Limbs	1	–	–	–	2	–	–	–	1	–	4	Operations 10.
Amputation of breast	–	–	1	–	–	–	–	–	–	–	1	
Removal of tumour	–	–	–	–	–	–	–	–	1	–	1	
Excision of joints	1	–	–	–	–	–	–	–	–	–	1	
Lithotomy	–	–	2	–	–	–	–	–	–	–	2	
Trephining	1	–	–	–	–	–	–	–	–	–	1	
Compound fractures	–	1	–	1	1	1	–	–	1	–	5	Injuries 18.
Incised wounds	–	–	–	–	–	1	–	–	–	–	1	
Punctured wounds	–	–	–	–	1	–	–	–	–	–	1	
Lacerated wounds	–	2	1	1	1	–	–	–	–	–	5	
Lupture of vein	–	–	1	–	–	–	–	–	–	–	1	
Urethral	–	–	–	–	–	1	–	–	1	–	2	
Disease of bones	–	1	–	–	–	–	–	–	–	–	1	Diseases 19.
Disease of joint	–	1	3	–	–	–	–	–	–	–	4	
Abscess	–	–	–	–	1	1	–	2	1	1	6	
Ulcer	–	–	1	–	–	–	–	–	–	–	1	
Carbuncle	–	–	–	1	–	–	–	–	–	–	1	
Gangrene	–	–	–	–	1	–	–	–	–	–	1	
Bedsore	1	–	–	–	–	–	–	–	–	–	1	
Piles	–	–	1	–	–	–	–	–	–	–	1	
Cancer	–	–	–	2	–	1	–	–	–	–	3	
Total	4	5	10	5	7	5	–	2	5	1	44	

After operations	10
„ injuries	13
„ diseases	19
„ urethrel	2
Total	44

Cases of Pyæmia and Septicæmia in St. Thomas's Hospital, 1869–78.*

Table I.

Males	58
Females	23
Sex not stated	44
	125

Table II.—Relation between the Number of Cases of Pyæmia and the Cases of Erysipelas in each Year.

——	1869	1870	1871	1872	1873	1874	1875	1 876	1877	1878	Total.
Pyæmia admitted	2	2	-	2	–	–	1	1	2	–	10
Pyæmia originating	2	2	7	17	16	13	15	11	20	12	115
Erysipelas admitted	†	1	1	37	60	†	†	25	29	†	153
Erysipelas originating	16	21	22	45	51	75	40	21	41	†	332

Table III.—Age in 117 Cases.

1–10	10–20	20–30	30–40	40–50	50–60	60–70	Total.
13	23	15	31	17	11	6	117

Table IV.—Average Duration of Diseases in 45 Cases.

	Days.
From admission to invasion	25·8
From invasion to death	6·2

* From "Hospital Reports."—C. H. R.
† Not stated.

Table V.—Details of the 124 Cases.

——	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	Total.
Amputation of limbs	–	–	2	1	3	4	4	5	7	1	27
Amputation of breast	–	–	1	1	1	–	–	–	–	–	3
Removal of tumours	–	–	–	1	1	1	–	–	–	–	3
Herniotomy	1	–	–	1	–	–	–	–	–	–	2
Osteotomy	–	–	1	–	–	–	–	–	1	–	2
Excision of joint	–	–	–	5	4	1	1	–	2	4	17
Incision into joint	–	–	1	–	–	1	1	–	–	–	3
Lithotomy	–	–	–	–	–	–	–	1	–	–	1
Ligature	–	–	–	1	1	–	–	–	–	–	2
Tapping	–	–	–	–	–	–	2	–	–	–	2
Incisions	–	–	–	–	–	–	1	–	1	–	2
Galvano Cautery	–	–	–	–	–	–	–	–	1	–	1
Compound fractures and dislocations.	–	–	1	1	2	2	1	1	1	–	9
Simple fractures	–	–	–	–	–	1	–	–	–	–	1
Lacerated wounds	–	1	–	–	1	–	–	–	1	–	3
Contused wounds	–	–	–	–	1	–	–	–	–	–	1
Urethral	1	–	–	1	–	–	3	–	2	–	7
Diseased bone	–	–	–	–	–	1	–	1	1	2	5
Diseased joints	–	1	–	1	–	1	–	–	–	1	4
Abscess	–	–	–	4	–	1	–	1	1	3	10
Ulcer	–	–	–	–	–	–	–	–	2	–	2
Fistula in ano	–	–	1	–	–	–	–	–	–	–	1
Various	–	–	–	–	2	–	2	1	–	1	6
Admitted pyæmic	2	2	–	2	–	–	1	1	2	–	10
Total	4	4	7	19	16	13	16	11	22	12	124

Pyæmia following operation	–	65
,, ,, injuries	–	14
,, ,, urethral	–	7
,, ,, diseases	–	29
,, ,, admitted	–	19

Details of Fatal Cases of Pyæmia and Septicæmia originating in University College Hospital, 1870–1878.*

Table 1.

Males	57
Females	21
Total	78

Table 2.

Year.	Jan.		Feb.		Mar.		Apr.		May.		June.		July.		Aug.		Sept.		Oct.		Nov.		Dec.		Total.
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
1871	1	–	1	–	3	–	–	1	–	–	–	1	–	–	–	–	–	–	–	2	–	–	1	–	10
1872	–	1	–	–	1	2	1	–	–	–	1	–	–	–	1	–	–	1	–	–	–	–	–	–	8
1873	1	1	–	–	1	–	1	–	–	–	–	–	–	–	–	–	–	–	1	–	1	–	–	–	6
1874	–	–	4	–	–	–	–	–	–	1	–	–	–	–	1	–	1	–	2	–	2	–	2	2	15
1875	–	–	1	1	2	1	1	1	1	–	–	–	1	–	–	–	–	–	–	–	1	–	–	–	10
1876	–	–	1	–	–	–	–	1	1	–	–	–	3	–	2	–	1	–	–	–	–	–	1	–	10
1877	–	–	1	–	1	–	1	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	5
1878	–	1	2	–	1	–	2	–	1	–	1	–	–	–	2	2	1	–	–	–	–	–	–	1	14
Total	5		11		12		9		4		3		4		8		4		6		5		7		78

Table 3.

	Days.
Average period of stay in hospital before development of disease in 55 cases	22·7
Average duration of disease from invasion to death in 60 cases	16·2

Table 4.—Details of 78 cases.

After Operations (23).

Amputations.	Excisions.	Lithotomy.	Trephining.
11	6	4	2

After Injuries (10).

Compound fractures or dislocations.	Fractured skull.	Simple fractures.	Wounds.
3	2	3	2

* From Annual Report of Registrars.—C. H. R.

During Progress of Disease (31).

Abscess.	Ulcer.	Whitlows.	Buboes.	Bedsores.	Cancer.
7	0	2	1	1	1

Empyæma.	Dis. of bone.	Dis. of joints.	Aneurism.	Various.
1	8	5	1	4

Urethral. 14

Pyæmia arising after operations	-	-	23
„ „ injuries	-	-	10
„ „ diseases	-	-	31
„ „ urethral	-	-	14
			78

TABLE 5.—RELATION BETWEEN THE CASES OF PYÆMIA OCCURRING IN THE HOSPITAL AND THE CASES ADMITTED, AND THE CASES OF ERYSIPELAS ADMITTED AND ORIGINATING IN EACH YEAR.

——	1871	1872	1873	1874	1875	1876	1877	1878
Pyæmia originating -	10	8	6	15	10	9	5	14
Pyæmia admitted -	2	–	1	3	1	1	–	–
Erysipelas originating -	28	9	19	35	16	45	13	*
Erysipelas admitted -	7	20	7	24	13	4	* 6	*

CASES OF DEATHS OF PYÆMIA AND SEPTICÆMIA OCCURRING IN ST. BARTHOLOMEW'S HOSPITAL FROM 1869–1878.†

Total number - - 50

RELATION BETWEEN CASES OF PYÆMIA ADMITTED AND CASES OCCURRING IN HOSPITAL AND THE CASES OF ERYSIPELAS.

——	1869	1870	1871	1872	1873	1874	1 75	1876	1877	1878
Pyæmia admitted -	3	2	5	2	2	3	4	4	1	2
Pyæmia occurring -	7	7	5	6	2	5	5	4	2	7
Erysipelas admitted	79	92	64	145	109	131	148	69	59	58
Erysipelas occurring	67	47	19	42	41	64	44	39	20	30

* Not ascertained.
† From the Annual Hospital Reports.

Deaths from Pyæmia and Septicæmia in the Seamen's Hospital (Dreadnought), Greenwich, 1869–1878.*

Table I.

Admitted with disease	7
Poisoning in hospital	53
	60

Table II.—Relation between Cases admitted and those arising in Hospital.

——	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	Total.
Cases admitted	–	1	1	–	–	1	1	3	–	–	7
„ arising in hospital	4	4	8	8	5	9	6	3	4	2	53
Total	4	5	9	8	5	10	7	6	4	2	60

Table III.

Pyæmia, septicæmia—arising after	
Operation in	21 cases.
Injury „	8 „
Disease „	29 „
Urethral affections	2 „
	60

* Compiled by W. J. Smith, Resident Surgeon, Seamen's Hospital.

Charles Henry Ralfe.

APPENDIX II.

TABLE I.—CASES OF DEATH FROM BLOOD-POISONING WITHOUT THE OCCURRENCE OF SECONDARY INFLAMMATIONS,—SEPTIC INTOXICATION AND SEPTIC INFECTION.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	F.	74	Extirpation of a large tumour from beneath the jaw (adenoma).	April 4 -	April 6 -	2 days	Admitted March 31st. Two days after operation she was very weak and ill. She had lost some blood, but not much, at the operation. Pulse intermittent. On the third day the dressings were removed; the wound was distended with offensive pus. In the afternoon there was dyspnœa, and she died in the evening. No rigors. No vomiting or diarrhœa.	Post-mortem twenty-four hours after death. Marked post-mortem staining of the endocardium. *Liver* and *spleen* pale and soft.
2	F.	36	Pelvic abscess after labour; varicose ulcer on leg; labour 5 weeks before admission.	Admitted Jan. 10, 1870.	Jan. 15, 1870	?	After labour the ulcer inflamed and extended. On admission ulcer very foul. Diarrhœa; sallow tint of skin; sweet smell of body and breath; delirium. Temperature not recorded. No history of rigors.	Post-mortem twenty-four hours after death. Extensive pelvic abscess; dark clots in pelvic veins. Marked post-mortem staining of endocardium. *Lungs* congested at bases. *Spleen* soft. *Liver* natural. *Kidneys* congested.
3	F.	6	Excision of ankle-joint.	Feb. 17, 1878 ?	Feb. 19, 1878	2 days ?	Patient admitted December 17th, 1877. Joint excised Feb. 16th. Became delirious at 2.30 p.m. on 18th. At 8.30 p.m. vomited. Temp. 96°. At midnight temp. 98°. Feb. 19th.—At 2.30 temp. 100°·4; 3.50 died. Temperature half an hour after death 98°·5.	Yellowish tint of skin. *Wound.*—Edges deep red; swelling of leg as high as knee; diffuse suppuration for one inch around the wound. Bones healthy. *Veins* healthy. *Glands* in groin swollen and commencing to suppurate. *Heart* healthy; no petechiæ. *Lungs.*—"Fluid like unaltered

								blood in the bronchi." Air vesicles in some places filled with similar fluid. Ecchymoses in submucous tissue of larger bronchi. Fauces and posterior nares contain altered blood. *Liver* firm. On section centre of lobules canary yellow; middle zone brick red; outer zone "translucent greyish colour." *Kidneys* swollen. *Spleen* small and firm. *Stomach* contained altered blood and mucus.
4	M.	65	Burn of arm; secondary amputation of arm.	Nov. 12, 1877	Nov. 16, 1877	4 days	Admitted October 17th, 1877. Whole of left arm severely burnt. 24th.—Amputation in middle of arm. The wound sloughed, and the discharge was very offensive. By November 6th the sloughs were separating and the stump cleaning. Temperature steady, about 100° to 101°. On the 12th he had a violent rigor. Temp. 105·2°. 13th.—Two rigors. The end of humerus could be felt bare. 14th.—Amputation at shoulder joint. 15th.—Two rigors. 16th.—One rigor. Temp. 103°. Bacteria were observed in the blood during life.	*Wound* somewhat foul. *Veins* healthy. *Lungs* healthy. *Heart* healthy. *Spleen* soft and swollen. *Liver* friable and swollen. No other abnormal appearances noted.
5	M.	17	Exostosis of femur; operation.	May 21, 1876	June 12, 1876	22 days	Tumour growing from condyle of femur. Removed May 20th. Rigor next day. Rigors repeated June 6th (2), 7th, 8th, 9th, 10th, 11th (2). Temperature irregular in the extreme; highest 106·8°; lowest 97·8°. Rising at time of death; 105·8°. Delirium.	*Wound* open. *Bone* bare, stripped of periosteum for lower two inches. *Knee-joint* full of pus, opening into wound. *Heart* flabby; 5 oz. of clear fluid in pericardium. *Lungs* congested. *Kidneys* congested. *Liver* normal. *Spleen* large and congested. No secondary inflammation anywhere.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
6	F.	47	Injury to knee; chronic disease of joint; amputation.	May 14, 1876	June 2, 1876	19 days	Old disease of knee resulting from injury. Open sinuses. Admitted April 24th. Knee removed by amputation, May 10th. 13th.—Temp. 102°. 14th.—104°; vomiting. 18th.—Sweats profusely; discharge healthy and abundant. 30th.—Severe rigor; not repeated. Died June 2nd.	Marked post-mortem staining. *Lungs* congested. *Kidney* fatty. *Spleen* soft and friable. No secondary abscesses anywhere.
7	M.	41	Disease of ankle-joint; amputation of foot.	?	July 13, 1876	?	Admitted April 26th for disease of ankle. Amputation of right foot on June 22nd. High fever on 23rd. Severe rigor on July 12th. Died on 13th.	*Lungs* congested; areas of grey colour. *Liver* oily. *Kidneys* fatty. *Veins* in thigh and leg above amputation were found full of firm coagulum, non-adherent.
8	M.	29	Lacerated and contused wound of forearm; compound fracture of radius.	?	July 21 18 6	Probably abont 2 weeks.	Admitted June 30th, 1876. Sloughing of wound; much swelling, requiring incisions; discharges decomposing. No rigors. Prolonged elevation of temperature; no great variations; highest temp. 104°; tongue gradually becoming dry; jaundiced tint of skin; no vomiting or diarrhœa. Respirations quick; 38 before death.	No secondary inflammations. *Lungs* congested; a little bloody fluid in one pleura. Nothing else noteworthy except that the *intestines* were greatly distended with gas.
9	M.	46	Compound fracture into ankle-joint; secondary amputation. Diffuse inflammation up leg; secondary amputation 14th day.	?	July 26, 1876	Probably about 10 days.	Sloughy condition of flaps; œdematous swelling of limb; foul discharge. Delirium; dry tongue; no marked vomiting or diarrhœa; no rigors; steady high temperature, about 104° to 105°. Respirations 42 before death. *Admitted July 6th, 1876.	No secondary inflammations. Hypostatic congestion of *lungs*. Marked post-mortem staining; ʒvj of blood-stained fluid in the *pleura*. *Spleen* pulpy. *Liver* soft; 60 oz. *Kidneys* slightly contracted. (This case was in the same hospital, but not in the same ward or under the same surgeon as the preceding.)

10	F.	70	Epithelioma of tongue; ligature of tumour.	June 14, 1871	June 19, 1871	5 days	Admitted June 8th. Ligature applied to a portion of tongue about as big as a shilling on June 10th. Three days after was feverish. Six days after operation hæmorrhage occurred; temp. 101·6°. Next day, 17th, was unconscious; purple patch over one elbow. 18th.—Skin sallow; inflammation of conjunctiva; great prostration. Death June 19th.	Skin sallow. Stump of tongue foul and sloughy. No secondary inflammations. *Subserous petechiæ*. *Spleen* very soft, almost diffluent. *Liver* and *kidneys* stained with blood pigment and swollen. *Lungs*, great hypostatic congestion.
11	M.	38	Compound fracture of leg; secondary amputation of leg.	May 20, 1878	May 24, 1878	4 days	Admitted May 1st. Small wound closed with collodion. At first all seemed to be doing well. Temperature slightly but not much elevated. By the 9th pus had formed, and the collodion was removed. On the 15th the leg was much swollen; temp. 108°; discharge abundant and offensive. Rigors on 21st and 22nd; temp. 105°. Amputation after second rigor. Next day temperature rose to 107·5°, and he died early on May 24th.	*Veins* healthy. Nothing found at post-mortem examination beyond very slight swelling of liver and kidneys.
12	M.	7	Disease of knee; excision.	Jan. 31, 1879	Feb. 4, 1879	4 days	Admitted January 21st. Slight heat about knee-joint, contracted, and tender. Temp. 99·2°, but apparently in excellent health. Excision January 30th. Treated antiseptically. No odour in discharge at any time. A good deal of blood lost after operation. Vomiting commenced next day, and continued till death. Temp. never above 101°; fell to 96·2° before death. Limb swelled. Patient became cold, cyanotic, and pulseless, and death took place on fifth day after operation.	Post-mortem absolutely negative with the exception of some slight swelling of liver, spleen, and kidneys.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
13	M.	26	Gangrene of leg; amputation.	Sept. 29, 1876	Oct. 7, 1876	8 days	Admitted Sept. 7th. Three days before was seized with numbness of left leg. On admission lower two thirds of leg gangrenous. Amputation below knee, September 20th, followed by high temperature. Rigors, September 29th, and again October 6th. Sloughing of stump; foul discharge. Delirium the only important symptom noted. Temp. high, and moderately regular. Died October 7th.	Post-mortem showed nothing but congestion of the lungs, swelling of liver, spleen, and kidneys. The gangrene was found to be due to an aneurism in the ham, which had undergone spontaneous cure.
14	F.	56	Poisoned wound of thumb.	About Jan. 23, 1872	Jan. 30, 1872	About a week.	Two weeks before admission a whitlow formed on right thumb. Incision at end of week. Inflammation spread to common sheath of flexors. No rigors. Admitted January 27th. Very prostrate; delirious; muttering; skin hot and moist; sloughing of right cornea; wound very foul. January 29th.—Temp. 105·5°; tongue dry; profuse diarrhœa; no vomiting; albumen in urine; icteric tint of skin. Comatose before death.	*Petechiæ* scattered over body. *Blood* firmly coagulated. No early decomposition. *Wound* very foul and sloughy. *Veins* healthy. *Heart*, subpericardial petechiæ; very marked post-mortem staining. *Lungs* intensely congested. *Liver* soft. *Spleen* large and pulpy. *Kidneys* swollen, soft, congested. *Intestines*.—A few scattered hæmorrhages in the mucous membrane. Extravasation of blood into cavity of arachnoid.
15	F.	34	Retained placenta after miscarriage.	?	Jan. 7, 1877	?	Rheumatic fever ten weeks before admission. Miscarried eight weeks before admission. Admitted January 3rd. Prostrate; tongue red; pulse week. Liver dulness increased; diarrhœa; delirium Temp. steady, between 100° and 101° Fahr.	Numerous petechiæ on skin. *Uterus*.—Os patulous; mucous membrane congested; soft jagged clot, probably placenta, at right fundus. *Lungs* congested. *Heart*, vegetation on *right* semilunar valves; considerable recent disease of valves on left side. *Spleen* soft; 15 oz. *Liver* congested; 68. *Kidneys* large and pale.

16	M.	36	Injury to arm; diffuse cellulitis; incisions.	?	Jan. 12, 1870	?	Fell in a fit on January 8th and injured arm; followed by diffuse cellulitis. Admitted January 11th. Arm much swelled; blebs. Died next day.	*Forearm.*—Subcutaneous tissue soaked in purulent serum. Red discoloration of outer side of leg. *Veins* healthy. *Lungs* congested. *Spleen* pale and soft. *Kidneys* pale, swollen. *No secondary inflammation anywhere.* Marked post-mortem staining. *Blood* fluid.
17	M.	54	Compound fracture of arm.	Mar. 18, 1875 ?	Mar. 21, 1875	3 days ?	Admitted March 17th. Restless. Violent delirium on the 18th. Died on the 21st.	*Lungs* œdematous; bronchitis. *Heart* uncontracted; marked blood staining. *Blood* fluid. *Liver* fatty. *Spleen* pulpy. *Kidneys* granular.
18	F.	26	Suppurating bursa patellæ.	April 27, 1876	May 1, 1876	4 days	Admitted April 26th. Abscess opened. Next day became feverish and delirious. Temp. 104·4°; pulse 150. Died rapidly.	*Lungs* congested. No secondary abscesses. *Liver* granular, tissue light yellow, soft, and oily. *Kidneys* greatly congested. *Spleen* not mentioned.
19	M.	53	Lacerated wound of wrist.	Jan. 7, 1878	Jan. 11, 1878	4 days	Admitted January 7th. Diffuse spreading inflammation commenced same day. The chief symptom noted was delirium. Notes imperfect.	Body well nourished. Extensive cellulitis extending up arm to axilla. *Veins* healthy. *Large* ante-mortem clot in right side of breast. *Liver* fatty. *Spleen* enlarged. *Lungs* congested. No petechiæ. *Other organs* healthy.
20	M.	38	Extravasation of urine (cause not mentioned).	Sept. 20, 1877	Sept. 24, 1877	4 days	Admitted on September 6th, 1877, with great extravasation of urine. Free incisions. On September 20th had a rigor; temp. 105°. Rigor repeated daily till day of death. Temperature high, irregular, never falling below 102°; highest 106°. Sweated profusely. Had some pain in calves and in one joint of little finger.	Extensive sloughy wound in perinæum. *Bladder* inflamed; "diphtheritic-like membrane" on mucous membrane. *Pelvis of kidney* slight recent inflammation; kidney swollen, pale streaks reaching to cortex; some points on the verge of suppuration. *Lungs* intensely congested. *Liver* fatty. *Spleen* 9½ oz., large, soft, and swollen.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
21	F.	46	Abscess in calf of leg.	Sept. 19, 1872	Sept. 27, 1872	8 days	Admitted September 16th, 1872, with a large abscess in calf; cause uncertain. Opened antiseptically. Dressing failed. Discharge offensive. Leg had been swelled for five weeks before admission. Old cured hip disease of opposite side. Unhealthy woman. Severe rigor on the 19th; temp. 105°. After that temperature persistently high, about 103°. Occasional vomiting; dry tongue; sallow tint of skin; gradually became weaker, and died; no diarrhœa.	Diffuse suppuration spreading from seat of primary abscess amongst muscles and subcutaneous cellular tissue as high as groin. *Knee* of same side full of pus. *Veins* healthy. *Lungs* congested. *Liver* large and soft. *Spleen* swollen and soft. *Kidneys* swollen and soft. No marked post-mortem staining.
				Date of Admission.	Date of Death.			
22	F.	26	Pin operation for varicose veins.	April 6, 1875	April 30, 1875	Dark adherent clot in the veins operated on. *Lungs* congested. *Liver* large and very soft. *Spleen* 9¾ oz., large and soft. *Kidneys* pale.		
23	M.	35	Suppuration beneath nail of r. thumb and middle finger (dissecting porter).	Jan. 18, 1875	Jan. 19, 1875	No cellulitis anywhere. No enlarged glands. Veins healthy. Blood dark and fluid. No bacteria seen in it. *Lungs*, some congestion and œdema. *Liver* soft. *Spleen* 9 oz., not particularly soft; greasy. *Kidneys* cloudy swelling; pale, and flabby. *Brain*, excessive vascularity of pia matter. No *petechiæ* anywhere.	
24	M.	75	Extravasation of urine.	April 19, 1877	April 26, 1877	Perineal section had been performed. *Lungs*, marked hypostatic congestion. Heart large and flabby. *Liver* normal. *Spleen* 18 oz., swollen, soft, decomposed. *Kidneys* enlarged and soft, cortex swollen.		
25	F.	34	Fibroid tumour of uterus; operation.	April 4, 1877	April 29, 1877	Post mortem twenty-eight hours after death. Yellowish tint of skin. *Uterus* enlarged. Free recent incisions in os. Sloughy mass of tumour in cavity of uterus. *Veins* healthy. All organs *decomposing*; bubbles of gas, &c. *Spleen* 11 oz., much enlarged. *Lungs* congested in dependent parts. Great post-mortem staining from decomposition.	
26	F.	40	Cellulitis of arm from poisoned wound; recent parturition.	Dec. 24, 1876	Dec. 26, 1876	Extensive cellulitis of arm. Lungs much congested; œdematous. Slight broncho-pneumonia of lower lobes. *Heart* flabby. *Liver* pale and soft. *Spleen* 9½ oz., soft, and pulpy, almost diffluent. *Kidneys* large and soft; cortex swollen.	

27	M.	33	Tumour of thigh; sloughing.	Dec. 25, 1876	Jan. 8, 1877	Post mortem twelve hours after death. Large sloughy tumour in front of thigh, extending down to femur. Muscles round about showed signs of inflammatory œdema. *Lungs*, slight hypostatic congestion. *Heart* soft and flabby. Endocardium not stained. *Liver* contained numerous bubbles of gas. *Spleen* 9½ oz., soft, and decomposing. *Kidneys* much decomposed; bubbles of gas.
28	F.	41	Pregnancy; dead fœtus; ovarian cyst; obstruction of intestine by pressure.	Mar. 6, 1877	Mar. 11, 1877	Slight commencing peritonitis. No fluid. Intestines enormously distended. Ovarian cyst 7 inches in diameter. A mass of blood clot below it. Descending colon narrowed. Uterus contains remains of fœtus (about third month) in a grumous mass. All the organs full of bubbles of gas, and too far decomposed to admit of examination. Post mortem thirty hours after death. Weather cold and frosty.

TABLE II.—PYÆMIA. GROUP 1 (*a*): CASES OF "PYÆMIA" IN WHICH NO THROMBOSIS, WITH SOFTENING OF THE THROMBUS WAS FOUND, AND IN WHICH NO ABSCESSES WERE PRESENT IN THE VISCERA.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	M.	46	Lacerated wound of forearm.	May 19 (?) 1874	May 29, 1874	About 10 days.	Wounded forearm with glass a month before admission. Admitted May 29th. Ten days before admission he complained of feeling very ill, and sick. Got worse. No rigor. Intense jaundice; dyspnœa; râles in chest. Liver two inches below ribs. Wound in arm dry and glazed. Various joints swollen. Died day after admission. Profuse sweating. Temp. 103°; resp. 50; pulse 148.	Large *open sore*, smooth and glazed. Left knee, right shoulder, right knuckles, contained excess of synovia, stained yellow. *Lungs* greatly congested. *Liver* 4½ lbs., large, firm, uniform dirty brown colour; bile ducts healthy. *Spleen* small, pale, and firm. *Kidneys* intensely congested and stained. *Veins* healthy.
2	M.	76	Whitlow; amputation of middle finger.	Mar. 25, 1875	Mar. 31, 1875	6 days	Whitlow formed a month ago. Amputation. March 20th, admitted into hospital. Did well till fifth day, when diffuse swelling and redness appeared on the hand. Temp. 99° to 100°. Abscess forming over elbow. Higher temp. 103° on 27th. Temp. at death 100°. No rigors.	No repair of wound; sloughy. *Veins* healthy. Abscess opening from elbow-joint of same side into subcutaneous tissue; diffuse cellulitis about elbow. *Lungs* congested. *Spleen* pale and firm. *Kidneys* fatty.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
3	F.	24	Strumous disease of ankle-joint; amputation of leg (left).	Feb. 27, 1875	Mar. 14, 1875	15 days	Disease of ankle about two and a half years. Admitted January 22nd, 1875. Amputation of leg, 27th. Stump slow in healing. Some diffuse inflammation. February 22nd.—It is described as granulating healthily. 27th.—A rigor; rigors repeated March 2nd. Pain in joints. Abscesses. Diarrhœa. Highest temp. recorded, 103°; moderately regular.	Emaciation. *Stump* unhealed. *Veins* healthy. Right knee full of pus; left knee excess of synovia. Right upper tibiofibular articulation destroyed. *Lungs.*—Pneumonia at base of right lung. *Spleen* small and firm. *Liver* congested and soft. *Kidneys* large and fatty. *Other viscera* healthy.
4	M.	6	Disease of hip -	Dec. 2, 1876	Dec. 17, 1876	15 days	Admitted for abscess forming over trochanter. Abscess opened and bone gouged away. Profuse suppuration. Steady high temperature. On December 2nd right shoulder, wrist, and ankle became swollen and painful. 4th.—Left ankle and sterno-clavicular joint. 13th.—Left knee. One slight rigor. 8th to 12th.—Profuse diarrhœa. Steady high temperature till death, averaging 103.	*Right hip.*—Head of femur necrosed; acetabulum carious; sinuses in all directions. *Joints.*—Left knee and ankle, right wrist, and both sterno-clavicular joints full of pus. *Veins* healthy. *Glands* under sterno-mastoids suppurating. *Spleen* healthy and firm. *Kidneys* swollen. *Other organs* healthy.
5	M.	26	Compound fracture of skull.	July 30, 1878	Sept. 18, 1878	49 days	Four days before admission cut his head. At first treated as an outpatient. Admitted July 30th, with wound very foul; slightly delirious. Urine free from albumen. Temp. varied from 102° to 104°. First rigor on August 3rd; repeated 4th. After this, although the temperature remained irregular and high (103° to	Wound unhealed. A small sequestrum partly separated at posterior part of occipital bone. A fracture about ¾ inch in length but united in the inner table. No burrowing of pus. Diploë healthy. *Joints.*—A very small quantity of viscid puriform fluid in the right acromio-clavicular and left wrist-joints. In

							99°), he had no further rigor till 14th (2), 15th (2), 16th, 18th, 19th. After this there was no rigor till Sept. 10th; repeated 15th and 17th. Temperature always remained slightly above normal; varying from 99° to 102°. On August 21st the right acromio-clavicular joint swelled; and on 24th the left wrist. Beyond this and the progressive emaciation there were no marked symptoms. On 29th profuse diarrhœa commenced, which lasted till death. Tongue became dry and brown. Emaciation excessive; he gradually died. Blood examined at various times, nothing found but granules of various sizes, and a gradually increasing deficiency of red and excess of white corpuscles. The wound granulated feebly.	the viscera, nothing was found except slight congestion of the intestinal mucous membrane. Slight swelling of the *spleen* and *kidneys*. *Other organs* perfectly healthy. Body extremely emaciated.
6	M.	47	Femoral aneurism; ligature of external iliac.	May 7, 18?	May 12 -	5 days	Aneurism ten months. Admitted April 13th. On 14th the external iliac was tied. 21st.—Greater part of wound closed by first intention. May 7th.—Became restless and uncomfortable. 9th. — Still restless, temp. 104°; tongue dry. 10th.—Delirious; pain in shoulder. 11th.—A rigor; pain in shoulder.	*Artery* healthily occluded. *Lungs* congested. *Liver* fatty. *Spleen* firm. *Kidneys* pale; tubules choked with epithlium. *Joints*.—Right hip and shoulder full of pus. *Blood* fluid twenty-five hours after *death*.
7	M.	50	Fracture of base of skull.	May 17, 1874	May 21, 1874	4 days	Admitted May 7th. Well, and to sit up 15th. Pain in left knee 17th; temp. 104. Exact line of fracture not stated. No rigor noted.	*Brain* healthy. *Viscera* normal. Left knee and right ankle full of pus. Serous effusion in intermuscular tissue of the arm and calf.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
8	F.	53	Chronic ulcer of leg	Sept. 10, 1874	Sept. 22, 1874	12 days	Admitted September 9th. 10th.—Great pain over inner condyle of left femur. 15th.—Temp. 103°. Pain in the knee and right elbow. 22nd.—Temp. 105°. Stupor. *Pustular* eruption on body.	Organs presented no particular abnormal appearance. *Veins* healthy. Both knee-joints contained excess of fluid. No pus.
9	M.	63	Stricture of urethra; simple dilatation.	July 23, 1876	Aug. 8, 1876	16 days	Stricture five years. Admitted July 19th, with retention. Simple dilatation. Rigors, July 23rd (2). Normal temperature for two days. Rigor July 27th, 28th, August 3rd, 4th (3), 5th. Tongue dry and brown. Occasional vomiting. Highest temp. 105°; temperature irregular. Began to complain of pain in joints on August 6th.	Stricture at bulb. A membraneous layer of lymph on the mucous membrane. Cystitis. No pyelitis. *Veins* healthy. *Joints.*—Left elbow and right knee contain pus. Some diffuse suppuration around flexor carpi radialis on right side. Great staining of viscera; early decomposition. *Liver* large and soft. *Spleen* 16 oz., very soft. *Kidneys* deeply congested. Some small hæmorrhagic spots.
10	M.	27	Removal of displaced cuboid bone; Syme's amputation.	July 25, 1875	April 27, 1875	62 days	Admitted December 29th, 1874. First operation set up general synovial disease of tarsus. Syme's amputation performed July 18th, 1875. 25th.—Temp. 104°. Stump sloughing; delirious. 27th.—Swelling appeared over right elbow: not connected with the joint. March 4th.—Abscess forming over right great trochanter. 16th.—Large abscess over external condyle of humerus. 30th.—Abscess forming on back of right shoulder. No fresh abscesses formed. He gradually became exhausted and died. Temperature moderately steady; about 103°. *Rigors* March 5th, April 24th (3). Occasional diarrhœa.	Body greatly emaciated. *The abscess* over the elbow had burst into the joint, but it had evidently commenced outside. The other abscesses were situated superficially. No post-mortem staining. No early decomposition. *Veins* healthy. *Liver* and *Spleen* albuminoid; *Spleen* not swollen. Cloudy swelling of *Kidney*. Other viscera healthy.

11	M.	36	Stricture of urethra; internal urethrotomy.	Oct. 12, 1874?	Oct. 23, 1874	11 days	Admitted October 9th. Retention. Internal urethrotomy 12th. *Rigor* soon after. During the next few days he had vomiting, headache, and thirst. Another rigor on 17th. 21st.—Very weak; delirious. 22nd.—A patch somewhat resembling phlegmonous erysipelas appeared on right forearm, and a similar patch on dorsum of right foot. Œdema and swelling of left arm. 23rd.—He died. Temperature steady, high, averaging 103°.	*Urethra.*—False passages; sloughy track. *Veins* healthy. *Kidneys.*—Acute interstitial nephritis of right kidney; cloudy swelling of left. *Other organs* healthy. Extensive patch of diffuse cellulitis in right arm; almost gangrenous.
12	M.	47	Stricture of urethra; internal urethrotomy.	May 14, 1878	June 13, 1878	30 days	Stricture of many years standing. Admitted April 3rd, 1878. In good general health. Internal urethrotomy May 11th. Rigor on the 13th. Rigors repeated frequently during this day; after that none recorded. There were frequent vomiting, progressive emaciation, and much cough. On the 23rd a large abscess formed on right side of chest. 27th.—Swelling of right knee. June 3rd.—Large superficial abscess at back of arm; tongue foul and dry; occasional profuse sweating. *Temperature.*—Highest recorded 103·6°; during the whole later period there was a marked evening rise; morning temperature nearly normal.	*Wound.*—Opening in the floor of urethra communicating with large perineal abscess. Prostate swollen. Contained many abscesses. *Lungs.*—Broncho-pneumonia of lower part of each lung, with slight pleurisy. *Spleen* 11 oz. *Liver* normal. *Kidneys* pale, and swollen surface of each kidney marked by a few small abscesses. *Joints.*—Much pus in right knee, pus in left sterno-clavicular articulation. *Subcutaneous tissue.*—Large abscess in posterior surface of right forearm. Diffuse suppuration on right side of chest extending down to latissimus dorsi. The abscesses in the kidney were probably due to extension of the disease from the bladder, and were not "pyæmic."

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
13	M.	21	Lithotomy - -	Mar. 9, 1878	Mar. 18, 1878	9 days	Admitted March 4th. Had suffered from stone some years; apparently in good health. Lithotomy (lateral) 6th; free bleeding. 9th.—A rigor; tongue furred; slight vomiting. Rigor not repeated. Temp. high, moderately regular, 102° to 103·5°. 11th.—Pain in back and loins. 12th.—Delirious. 13th.—Excessive delirium. 14th.—Subcutaneous, brawny, inflammation patch over elbow. 17th.—This has extended some inches; similar patch over malleolus; tongue dry; no diarrhœa; no vomiting. *Blood* examined, nothing abnormal found.	*Slight emaciation.* *Wound* sloughy. *Veins* healthy. *Glands* slightly swollen. *Spleen* soft, one softening infarct. *Kidneys* swollen. *Other organs* healthy. Over right elbow, on left forearm, and over right internal malleolus, and dorsum of foot are patches of diffuse suppuration in the subcutaneous tissue. Diffuse cellulitis in front of larynx, beneath deep fascia. *Blood* imperfectly coagulated; no organisms recognised in it.
14	M	18	Lithotomy - -	Sept. 13, 1877	Oct. 2, 1877	19 days	Admitted August 22nd. Tight traumatic stricture with medium-sized phosphatic calculus in bladder. Lateral lithotomy September 4th. Considerable hæmorrhage; wound of rectum. Went on tolerably well till the 12th; secondary hæmorrhage. 13th.—A violent rigor. 16th.—Acute pleuritic pain on left side. Effusion abundant by the 19th. 20th.—Signs of hydropneumothorax; only one rigor; persistent high temperature, somewhat irregular, varying from 100° to 104°; no diarrhœa; no vomiting; rapid emaciation; complained of pain for a few days (19th to 22nd) in acromio-clavicular joint.	*Wound* foul and sloughy. *Veins* healthy. *Glands* at brim of pelvis swollen. *Heart* healthy, no post-mortem staining. *Left pleura* contained 48 oz. of offensive pus and some gas, the pus teeming with bacteria (17 hours after death). *Lung* compressed, but otherwise healthy. *Right lung* healthy. *Liver* and *Spleen* perfectly normal. *Kidney.*—Slight pyelitis on both sides; slightly swollen. *Blood.*—Rouleaux imperfect; excess of white corpuscles, no bacteria seen.

15	M.	45	Wound of arm; cellulitis.	Dec. 7, 1874	Dec. 13, 1874	6 days	Admitted December 4th. Cellulitis commenced on the 7th; bronchitis. 8th.—Cellulitis less; wound discharging freely. 10th.—Restless, delirious; rapid weak pulse. Death on the 13th. No rigors noted.	*Wound* sloughy; pus infiltrating intermuscular cellular tissue. *Viscera* presented nothing of interest. Small quantity of dark blood clot in great omentum. *Peritoneum* generally vascular and coated with a thin layer of lymph.
16	M.	51	Simple fracture of leg; suppuration and sloughing; amputation below knee; re-amputation at knee; resection of end of bone.	?	Feb. 25, 1874	—	Admitted October 24th, 1873, with a simple fracture of tibia. The skin sloughed, and the fracture became compound. Antiseptic dressing at first employed, but abandoned afterwards. On December 21st the leg was amputated. The flaps sloughed, and the patient was left with a large raw surface, and pus burrowing up the limb; copious offensive discharge; patient much emaciated and weakened. Temperature was not high, seldom above 100°. On January 17th he had a slight rigor, but on the 18th and 19th temperature was normal. On 21st, 100°. On 24th, normal; amputation through knee; an abscess in the thigh laid open, which had been forming for some weeks. Patient very weak; sweats profusely. Flap sloughing; temp. 100°; end of bone protruding. February 19th.—End of femur sawn off; flaps readjusted. 20th. — Temp. 102°. 21st, 22nd, 23rd.—About 100°. 24th.—Temp. normal; stump swollen; pain in thigh; no other symptoms, in fact, he seemed better, when he died suddenly, July 25th, 1874.	*Stump.*—No union; slightly sloughy. *Veins* healthy. *Lungs* slightly congested. *Abdominal viscera* and *brain* healthy. *Heart.*—Pericardium contained 2 oz. of pale yellow, turbid fluid; surface of heart injected. Muscular substance showed irregular polyhedral areas of pale yellow, central parts red, varying in size, mostly towards the pericardial surface. No subpericardial petechiæ. The patches were slightly softer than natural. Microscopic examination showed abundant cell infiltration between the muscular fibres. Fibres granular, striation imperfect in the inflamed areas. Endocardium healthy.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
17	M.	30	Whitlow - -	Dec. 1	Dec. 6, 1873	5 days	Cut his finger, November 21st. Swelling and pain, on the 22nd. On the 27th an incision was made at the root of the second finger; hand swelled, but patient did not seem particularly ill. Went out walking, December 1st, and found he had pain in right thigh. 3rd. Very weak and ill; restless; temp. 101°; pulse 80; tongue dry. Middle finger swollen; discharge very offensive; great swelling and œdema of right thigh; glands in groin enlarged. Admitted into hospital. 4th.—Temp. 103°. 5th.—Temp. 104°. 6th.—Temp. 101°; furred, dry tongue; very ill; no rigors; no diarrhœa. 6th.—Pericardial friction; dyspnœa; large abscess opened in thigh. Died.	*Finger.*—Suppuration in sheath of tendon; meta carpo-phalangeal joint open. *Veins* and *glands* healthy. *Thigh.*—Large foul abscess beneath deep fascia, no sharp limit to it. *Heart.*—Pericardium contains 2 oz. of turbid fluid. Serous surfaces roughened, but not much lymph. Valves reddened, chiefly from post-mortem staining. Vascular injection of aortic and mitral. Muscular substance dirty muddy colour throughout; in many parts broken down into a pulp. Hæmorrhages everywhere scattered. Microscopic examination showed abundant cell infiltration; fibres breaking down. *Lungs* gorged with blood. *Spleen* large and soft. *Kidneys* swollen.
18	M.	25	Fracture of lower jaw; compound fracture of three ribs, left clavicle, left femur, and left humerus; contused wound of hand.	About Oct. 1, 1872.	Nov. 21, 1872	52 days	Admitted September 26th. October 1st.—High temperature and delirium. Delirium continued more or less till death; tongue dry. 9th.—Profuse sweating; sudamina; offensive breath. Suppuration round the simple fracture of the thigh on the 20th. Pus drawn off on the 24th; not offensive; no organisms recognised in it. 29th.—Simple fracture of humerus suppurating. 30th.—Rib fracture suppurating also; great emaciation; continuous delirium. Temp. record imperfect,	Body greatly emaciated. Beyond the suppuration round the fracture nothing abnormal was found. All the viscera were perfectly healthy. No suppuration in *joints*. *Veins* healthy. Large bed sore ove crum.

							but after November 1st it was scarcely above normal. He gradually became weaker, and died on the 21st exhausted.	
19	M.	4	Stricture of urethra; perineal section.	July 17	July 27	10 days	Admitted July 12th. Stricture took No. 8. Perineal abscess opened on the 12th. 17th.—A rigor. 21st.—Perineal section. 22nd.—Slight shivers. 23rd.—Slight shivers; hiccough; delirium. 24th.—Tongue dry; slight shivers; vomiting; hiccough. 25th.—Vomiting; diarrhœa. 26th.—Diarrhœa; red blush on right middle finger; delirium; hiccough; vomiting. 27th.—Similar erysipelatous blush on left arm and forehead; constant hiccough and diarrhœa. He died. Temperature moderately regular, highest 105°; 103° at death.	*Wound* apparently healthy; numerous extravasation in sub-mucous tissue of bladder. Some small abscesses in the prostate. *Superficial ecchymosis* on left foot, left leg and thigh, and back of hand. *Kidney*.—Cortex swollen, red colour. *Spleen* very large and red. *Liver* very large. *Heart*.—Marked staining. *Lungs*.—Some broncho-pneumonia; old cavity at apex; some sub-pleural petechiæ. The swelling on the forehead when cut into looked as if infiltrated with pus, but there was no actual abscess. The viscera were examined microscopically (see Appendix IV.).
				Date of Admission.	Date of Death.			
20	M.	43	Disease of wrist; extensive suppuration up arm.	Mar. 24, 1875	May 13, 1875	Eight incised wounds in arm. *Right pleura*, $2\frac{1}{2}$ pints of semi-purulent fluid; *left*, 3 pints. *Lungs* healthy. *Liver* large, fatty. *Spleen* 4 oz., dark and soft. *Right knee-joint* contained a large quantity of pus and flakes of lymph. *Veins* healthy.		
21	M.	8	Primary amputation of right thigh; secondary hæmorrhage; ligature of common femoral.	April 4, 1876	April 23, 1876	Emaciated. *Wound* foul, sloughy. End of bone exposed, necrosed, with "appearance of osteo-myelitis." *Glands* in groin swollen. *Veins* healthy. *Joints*.—Left knee full of pus, apparently from an abscess which had commenced outside it. Effusion about left ankle; no pus. *Lungs*.—Grey granulations in pleuræ and lungs; some caseous patches; some small pyæmic infarcts. *Spleen* small and firm. No tubercle or other pathological changes in any other organ. (The infarcts in the lung seem to have been somewhat doubtful.)	

No.	Sex.	Age.	Primary Disease.	Date of Admission.	Date of Death.	Post-mortem Appearances.
22	M.	59	Iliac abscess from perityphlitis (?).	Feb. 1, 1876	April 2, 1876	Two sinuses opening into a large abscess cavity partly healed and granulating, and communicating with the cæcum. *Lungs* congested posteriorly. *Heart.*—Muscular substance pale and friable. *Spleen* large, very soft, pale and greasy. *Kidneys* large, intensely congested. *Brain.*—Pia mater filled with semi-purulent exudation; grey matter injected.
23	M.	14	Incised wound penetrating knee.	April 30, 1877	May 1, 1877	The wound penetrates the joint by a small opening; joint contains turbid semi-purulent fluid stained with blood; swelling and œdema of tissues outside the joint; edges of wound bathed in pus. *Veins* healthy. *Blood* in heart imperfectly coagulated, dark, treacly. *Spleen* 4 oz., slightly swollen. *Pancreas* injected. *Kidneys* gorged with blood. *Lungs* engorged with blood, with patches very dark in colour, almost hæmorrhagic. *Brain.*—Convolutions flattened; pia mater rosy in tint; slight sticky exudation at base.
24	M.	34	Abscesses connected with bladder.	Jan. 29, 1878	Feb. 28, 1878	Emaciation; slight recent *pericarditis*; slight recent *pleurisy*. *Lungs* healthy. *Heart* healthy. *Liver* apparently swollen. *Kidneys* healthy; an abscess below the pubes lying between the peritoneum and abdominal walls containing very foul pus.

TABLE III.—PYÆMIA. GROUP I (*b*): CASES OF PYÆMIA IN WHICH NO THROMBOSIS WITH SOFTENING OF THE THROMBUS WAS FOUND AS A SOURCE OF EMBOLISM, BUT IN WHICH VISCERAL ABSCESSES WERE PRESENT.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	M.	35	Compound fracture of right tibia, and left tibia in two places; secondary amputation.	July 12, 1877	July 20, 1877	8 days	Admitted June 21st. Wound in right leg healed under collodion; lower wound in left leg also covered with collodion. Suppurated on tenth day. Upper wound dressed with water dressing; suppurated freely; discharge foul. On July 12th had a severe rigor. Temp. 107·6°. 13th.—	Wound showed no sign of healing. No granulations. *Bones* healthy. *Veins* healthy. No petechiæ. *Lungs.*—Numerous "pyæmic" abscesses in upper part of lower lobe of each lung. Pleurisy localised over the abscesses. *Liver* slightly swollen. *Kidneys* swollen. *Spleen* slightly swollen;

							Rigor. 14th.—Amputation in lower one third of thigh. No rigor till July 17th, after that repeated rigors; sweating; diarrhœa; no vomiting; delirium. Temp. irregular, falling to normal after rigors; 103° at death. No post-mortem rise.	soft; not pulpy. *Other organs* healthy. *Blood* contained a few doubtful micrococci, not in groups.
2	M.	30	Stricture of urethra; simple dilatation.	?	May 3, 1875	—	Admitted January 23rd. Had repeated rigors from the time of admission to ten days before death. Notes of other symptoms imperfect.	Short false passage. Recent cystitis. Prostatic *veins* healthy. *Lungs.*—Right contains several pyæmic abscesses. Left collapsed, contains a small abscess. Left pleura full of offensive pus and gas. *Liver* and *spleen* soft and pulpy. *Kidneys* swollen.
3	M.	54	Fracture of patella and right olecranon.		Dec. 22 -	--	Fracture simple on admission, November 29th. On December 2nd the integuments over the fractured patella were very thin. 4th.—The knee was opened by sloughing. 5th —Erysipelas. 7th.—Erysipelas extended to trunk. 9th.—Suppuration at back of thigh; incisions. 12th.—The injured elbow has suppurated; incised; sloughing about wound; profuse sweating and diarrhœa. One rigor on the 20th. Temp. high, 104°, moderately regular.	Extensive abscesses burrowing amongst the muscles around the injured joints. "Secondary abscesses" in both lungs. Other organs healthy. *Veins* healthy.
4	M.	36	Wound of rectum by enema syringe.	Oct. 12 ?	Oct. 14, 1871	2 (?) days	Admitted for fracture of skull and laceration of brain. On October 7th, struggled violently during administration of an enema. No rigors. Temp. 103°. Dry tongue. Typhoid condition ending in death.	Foul abscess between bladder and rectum. Wound of rectum. Local peritonitis. *Veins* healthy. *Lungs.*—One infarct in left lung. Both lungs congested. Other organs healthy. Extensive fracture of skull and laceration of brain.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
5	M.	66	Stricture of urethra; cystitis.	?	Aug. 8, 1878	?	Patient stupid, and could give no account of himself. Notes imperfect.	Stricture about three inches down urethra. Short false passage. *Lungs.*—Right contains numerous collections of pus. Right *pleura* contains thin puriform fluid. *Spleen* pulpy. *Liver* fatty. *Bladder.* — Recent cystitis. Blood fluid forty-one hours after death.
6	M.	5	Disease of hip; old excision.	?	Jan. 29, 1878	?	Sinuses about hip enlarged. January 16th.—No bone removed. 25th.—Child much weaker. 29th.—Died. Notes imperfect.	Numerous sinuses leading to extensive caries of the pelvic bones. Foul pus. *Veins* healthy. *Lungs.*—Numerous "pyæmic abscesses" in both lungs. Some pleurisy over them. *Liver.*—Several pyæmic abscesses on surface. Peritonitis over them. Branches of the hepatic vein were found filled with softened clot. In the area from which these veins came were scattered points of suppuration. *Spleen* not enlarged. Other organs healthy, with the exception of a slight degree of lardaceous degeneration.
7	F.	54	Phlegmonous erysipelas of arm.	Nov. 29 -	Dec. 4, 1874	5 days -	Admitted November 20th, 1874. Cut her arm with glass some few days before. Pus burrowing amongst the muscles. Great swelling. 29th.—Free incisions. Rigor the same day. Rigor repeated November 30th, 31st, and December 1st and 2nd, not after. Discharge from wound offensive; much sweating; pain in chest; no diarrhœa or vomiting; no jaundice. Temp. irregular, highest 106°.	Sinuses burrowing in various directions. Discharge foul. *Veins* healthy. *Lungs.*—Right pleura contains recent lymph. Right lung, about fifteen "abscesses." Left side healthy. *Liver* contained innumerable abscesses, varying from a few minims to one ounce in capacity, surrounded by a zone of hyperæmia. Pus healthy and yellow. *Spleen* large and soft. *Kidneys* swollen.

8	M.	37	Operation for piles	Oct. 6	-	Oct. 17	-	11 days	Admitted September 23rd. Operation 28th. Rigor October 6th; frequently repeated; pain in chest; vomiting. Highest temp., 106°, on the day before death.	Slight jaundice. Empyema. *Lungs* congested. Secondary deposits. *Heart* uncontracted. Walls blood-stained. *Liver.*—Numerous pyæmic abscesses. *Veins* of lower part of rectum slightly dilated, containing dark red clot. No softened clot found.
9	M.	19	Disease of metatarsus; amputation; Lisfrançs.	Nov. 27	-	Dec. 9	-	12 days	Admitted November 23rd. Amputation 24th. Rigor 27th, repeated 28th. Stump red, swollen. December 2nd. —Redness has extended up leg; lymphatic streaks; small abscess in line of lymphatics; jaundice; vomiting. Rapid respiration. Death.	*Wound.*—Inner half nearly healed. Outer half foul and sloughy. Numerous small abscesses in skin of leg. *Veins.*—Dark healthy clot. *Lungs.* — Numerous secondary abscesses. *Liver.*—100 ozs. One or two abscesses. Tissue pale. *Spleen* soft and pale. One large abscess. *Kidneys* flabby, smooth; cloudy swelling. *Blood* fluid thirty-one hours after death.
10	F.	9	Disease of hip-joint	May 20	-	May 31, 1874		10 days	Admitted August 20th, 1873, with sinuses leading into hip-joint. Various incisions were made from time to time, and gouging operations performed, last on May 7th. On the 10th, a diffuse inflammation, resembling erysipelas, attacked the wound, with great œdema. This subsided somewhat by the 19th. On the 20th the leg began to swell greatly; temperature rose; swelling also in opposite leg; delirium; diarrhœa; much albumen in urine. One rigor on the 28th. Temperature fell before death.	The tissues for a considerable distance round the diseased hip are infiltrated with serum. *Veins* healthy. *Lungs.* — Numerous "pyæmic abscesses." *Liver.* — Several small abscesses. Glands in hilus swollen. *Kidney.*— Right, two abscesses. All abdominal organs present signs of very advanced albuminoid degeneration.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
11	M.	54	Disease of ankle-joint; incision into abscess, &c.	Feb. 12, 1877	Feb. 22, 1877	10 days	General health feeble; great swelling of ankle; incision made into joint. Admitted January 31st. Some further operation February 8th. Feb. 12th.—Temp. 103·4°; delirium; temperature continued high; tongue brown and dry; no attempt at union; great quantities of pus; sweating profusely.	Considerable emaciation. Imperfect union of soft parts. Bones bathed in pus. *Heart.*—Much blood staining. Fatty. *Lungs.*—Several recent deposits in lung-substance in both lungs. *Liver* congested. *Spleen* 10 oz., pulpy. *Kidneys* granular. *Brain.*—Two purulent deposits in right hemisphere, a patch of intense congestion in left. *Blood* very fluid twenty-seven hours after death.
12	M.	33	Abscess in chest walls.	July 22 (?) 1876	July 29, 1876	About a week.	July 13th.—Was feverish. 17th.—Pain in middle chest; swelling formed. 25th—It was lanced. 26th.—Admitted into hospital; abundant pustular eruption over body and limbs; skin in epigastrium, livid and mottled. Gaping wound over seventh and eighth ribs; foul and sloughy; dusky red; boggy. Swelling in right fore-arm, and another near right elbow. Pneumonia. Temperature 104°. Diarrhœa; delirium; increased prostration. Death three days after admission. Temperature persistently high till death.	*Wound* foul. No post-mortem staining. *Lungs* intensely congested. In anterior edge of each lower lobe is one recent infarct. *Liver* tough, not congested. *Spleen* soft and swollen, pulpy. *Kidney.*—Cloudy swelling. *Veins* healthy.
13	M.	16	Disease of ankle-joint.	Feb. 19	Feb. 24	5 days	Old disease of ankle. General health good. Various gouging operations. Admitted January 14th. Four days after last operation; on February 19th had a rigor. Abscess formed over elbow. Pneumonia; delirium. Death on the 24th.	*Wound* sloughy. Joint extensively diseased. Tibia inflamed as far as medullary canal. *Heart.*—Blood staining. *Lungs.*—Secondary abscesses. *Liver, spleen,* and *kidney* contained throughout substance bubbles of gas of decomposition. Abscesses in thoracic walls on left side, under tendon of triceps of arm. Diffuse cellulitis of upper two thirds of forearm. Post-mortem fourteen hours after death. Weather cold.

14	M.	21	Diffuse cellulitis of of arm.	July 25 -	Aug. 9 -	15 days	Admitted August 1st. A week before had pain in axilla. There had been a small sore on left forefinger. Pain increased; rigors; swelling of axillary glands. On admission tongue brown and dry. Pulse 100; respiration rapid; œdema and redness at inner side of left arm below the left elbow. 3rd.—Pneumonia. 4th.—Worse, both arm and lung. 7th.—Joints swelled. 8th.—Dyspnœa.	Post mortem sixteen hours and a half after death. Advanced decomposition. Extensive diffuse cellulitis of forearm. No wound. *Heart.*—Much blood staining. *Lungs.*—One small patch of secondary pneumonia, suppurating. Pneumonia of both bases. *Joints.*—Pus in right hip and knee. *Blood* very fluid.
15	M.	58	Stricture of urethra.	Jan. 20 -	Feb. 8 -	19 days	Stricture twenty years. Admitted January 5th. 19th.—Forcible dilatation, followed by rigors and vomiting. Small abscess formed in front of scrotum and was opened. 31st.—Rigors and constant vomiting continued. February 4th.—Hypogastric pain; swelling over right elbow; pain in shoulder.	*Urethra* showed several false passages. Jaundiced tint of skin. Great emaciation. *Lungs.*—Seconday abscesses in both lower lobes. *Liver.*—Large secondary abscess. *Spleen* soft and pulpy. *Kidneys* large and flabby. Pyramids congested. *Joint* not opened after death.
16	M.	50	Tubercular disease of testicle; castration.	Nov. 4, 1878	Nov. 11, 1878	5 days	Admitted October 22nd. Testicle enlarged six months. Castration November 2nd. Wound nearly healed by the 4th; in the evening a severe rigor. 5th.—A rigor. 7th.—Hæmorrhage from wound; cough; another rigor. 11th.—Died. Temperature during rigors 105·8°; at death 101°; morning before death 98·2°.	*Wound* dry and sloughy. Cut section of cord swollen, hard, nodular. Exudes pus from minute abscesses. *Heart.*—Numerous subpericardial petechiæ. *Lungs.*—Left pleura, half a pint of pus. Yellow lymph. Left lung, lower lobe studded with minute abscesses. On section, lower lobe dark red colour, solid, flabby, non-crepitant; large, old cavity at base. Right lung, numerous small abscesses. One or two dark patches near surface. Other organs healthy. Nothing abnormal in other organs.

No.	Sex.	Age.	Primary Disease.	Date of Admission.	Date of Death.	Post-mortem Appearances.
17	M.	53	Disease of finger. Amputation of right index finger.	May 12, 1875	May 16, 1875	Post mortem twenty-four hours after death. Right epitrochliar *gland* swollen and suppurating. *Veins* normal. *Lungs* œdematous and congested. *Heart* flabby and friable. *Liver* fatty. An abscess, size of walnut, on anterior surface. *Spleen* 10½ oz., soft. *Kidneys* congested, soft, and friable. *Brain.*—Excess of subarachnoid fluid. *Parotid gland* red and swollen, not suppurating.
18	F.	50	Diffuse abscess in thigh. Incisions.	Jan. 24, 1876	Feb. 2, 1876	*Wounds* and abscess cavity foul. Abscess had recently opened into the knee. *Glands* in groin swelled, not suppurating. *Lungs* deeply congested and œdematous. Bronchi inflamed. *Liver* small, otherwise healthy. *Spleen* 10 oz. Numerous infarcts softening in centre. Substance very soft. *Kidneys.*—Numerous points of intense injection with central suppuration.
19	M.	44	Amputation of left thigh.	April 3, 1877	April 25, 1877	Wound unhealed. *Veins* healthy. *Pleuræ.*—Recent lymph on both sides. *Lungs.*—Numerous softening infarcts in the lower lobes of both lungs. *Spleen* 8 oz., slightly enlarged, soft. *Liver* soft. *Kidneys* swollen. *Stomach.*—Minute erosions, and patches of hæmorrhages in mucous membrane. Infiltration of pus around pharynx and amongst the muscles at the root of the tongue.
20	M.	17	Diffuse cellulitis of neck and mediastinum, with suppuration. Incisions.	Dec. 7, 1875	Jan. 8, 1876	*Wounds* foul. Large area of diffuse suppuration extending from lower jaw to mediastinum. *Veins* healthy. *Lungs.*—Lower and middle lobes of right lung, solid and numerous "pyæmic abscesses" scattered through the solid area. Left in the same state. *Liver* pale. *Spleen* 6½ oz., large, pale, soft. Nothing else noticed.
21	F.	72	Spindle-celled sarcoma of thigh. Operation.	July 30, 1875	Mar. 26, 1876	Large scar in groin, with one part not healed, covered with smooth, glazed granulations. *Lungs.*—A branch of pulmonary artery contains a firm thrombus, with a small abscess in the corresponding part of the lung. *Liver* fatty. *Spleen* 6 oz., swollen, very soft, greasy.
22	F.	18	Chronic abscess of sacral region.	April 3, 1877	April 29, 1877	Large open abscess over sacrum. Caries of left side of sacrum. *Veins.*—No disease of veins found. *Pleuræ.*—Right, three pints of fluid, sero-purulent. *Lungs.*—Many patches of an irregular triangular form in lower lobe, probably not pyæmic. In left lung one "typical pyæmic abscess." *Liver* pale and soft. *Spleen* 7 oz., soft and swollen. *Kidneys* soft and swollen.

23	M.	23	Disease of right internal ear.	May 26, 1877	May 26, 1877	Mastoid cells contain thick cheesy matter and pus. Membrane tympani perforated, signs of chronic otitis media. Dura mater over petrous portion of temporal discoloured and thickened, loose. Roof of tympanum rough, pus oozes through on pressing on external meatus. Layer of lymph on arachnoid surface of dura mater over temporal bone. Sinuses healthy. *Brain.*—Large abscess in right hemisphere of cerebellum, almost entirely filling it. This contains no bacteria. Foul odour to pus. *Spleen* large, rather soft, deep red colour. *Kidneys* large, swollen.
24	F.	53	Sinuses in right groin and left lumbar region. Caries of vertebræ.	Nov. 14 -	Feb. 2 -	*Lungs* congested. Patches of "caseous deposit" scattered here and there in both lower lobes. *Liver* soft and swollen. *Spleen* 9¾ oz., soft. Several small abscesses about the size of a pea. *Kidneys* 14 oz., swollen.
25	F.	14	Disease of hip -	Feb. 12, 1875	Feb. 19, 1875	Body well nourished. Left thigh fuller than right. Purpuric blotches on skin of legs. *Joints*—Excess of fluid in left knee, slightly turbid. *Muscles* of thigh.—Small abscess in rectus femoris. Effusion of serous fluid amongst the muscles. *Left hip-joint* full of flocculent, bloody pus. *Bones* healthy. *Veins* healthy. *Pleuræ.*—Very slight pleurisy on right side. *Lungs* much collapsed. No infarcts. *Pericardium.*—Two ounces bloody, flocculent fluid. Recent slight inflammation. *Heart.*—Small abscess in wall of left ventricle. Small patches of injection and ecchymosis in substance of heart. *Liver.*—Scattered on surface are small yellow spots surrounded by a zone of injection. A few of these are softening. A few larger ones contain puriform fluid. *Spleen* 8 oz., soft, red. Several large infarcts. Some softening. *Kidneys.*—Numerous small infarcts in cortex. *Stomach.*—Slight ecchymosis.
26	M.	24	Ulceration of larynx	Aug. 16, 1878	Aug. 23, 1878	Old scars on various parts of body, one of excision of right shoulder. Recent tracheotomy wound. *Lungs.*—Numerous abscesses in lower lobe of left lung. One or two in right lung. *Heart* flabby. Marked staining of endocardium. *Larynx.*—Some signs of slight recent ulceration. *Liver* large and pale. *Spleen* large and soft, very vascular. *Kidneys* small and tough. A large *abscess* was between the brim of the pelvis and the bladder. Grumous curdy pus in *right* elbow. Bones healthy.
27	F.	72	Ulcer of ileum -	April, 1877	July 4, 1877	Old pleurisy on left side. *Lungs.*—Caseous pneumonia. *Pericardium.*—Slight commencing general pericarditis. *Heart.*—Recent warty vegetation on valves of left side. *Peritoneum.*—Slight peritonitis in lower part of cavity. Intestines in right iliac fossa matted together. Small perforating ulcer in ilium, a few inches from the ileo-colic valve. Another small ulcer a little lower down. Suppuration in mesenteric glands. *Large intestine.*—One or two small ulcers. *Liver* small. *Spleen* small and soft. *Kidneys.*—Great dilatation of calyces. Pyramids destroyed. Cortex thinned. *Ureters* dilated. *Uterus* slightly enlarged. Cavity contains bloody fluid. *Vagina.*—Ulceration at upper part. A small collection of pus behind the pharynx.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Post-mortem Appearances.
28	M.	36	Disease of hip-joint	Feb. 27, 1877	Mar. 18, 1877	Great emaciation. Large, foul, suppurating cavity where the head had been excised. Upper two inches of shaft necrosed. Periosteum separated from bone to within three inches of knee. Medulla infiltrated with pus. Abscesses burrowing into pelvis and gluteal region. *Pleuræ*.—Recent pleurisy over lower lobes of left lung. *Lungs*.—Numerous infarcts breaking down in centre. Numerous grey granulations. These are entirely free from opacity, and are uniform in size. *Liver*.—One grey granulation in capsule. *Spleen* 2½ oz., slightly enlarged. *Kidneys*.—Cortex pale. *Brain*.—On surface, beneath pia mater, are numerous yellowish patches, rounded mostly, not prominent, varying in size from a millet seed to a pea. On section containing puriform fluid. Each hemisphere contains a large caseous mass, the size of a walnut.

TABLE IV.—PYÆMIA.—GROUP II.: CASES IN WHICH THROMBOSIS OCCURRED APPARENTLY AS A SECONDARY EFFECT OF BLOOD POISONING.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	M.	66	Primary amputation at wrist.	June 16, 1875	July 1, 1875	15 days	Amputation April 29th. May 10th.—All going on well. 22nd.—Suppuration; pus squeezed from between flaps; swollen up to elbow. 24th.—Swelling continues. 30th.—Incisions; abundant suppuration; end of radius protruded from stump. June 16th.—First rigor. Rigors June 17th, 18th, 19th, 23rd, 25th, 26th, 30th (2). Irregular temperature, highest recorded 104°. Progressive emaciation and weakness the only symptoms noted. Notes imperfect.	Considerable emaciation; œdema of both legs. *Stump*.—End of radius sticks out; abscess amongst muscles of forearm. Both *bones* more or less extensively necrosed. *Veins* healthy in forearm and arm. *Large abscess* in and under right soleus; popliteal vein opened into abscess and was distended with pus; this reached as high as Poupart's ligament, where there was a small, firmly-adherent, laminated clot. Pus in small veins opening into femoral and popliteal. *Lungs*.—One embolic abscess. *Liver* pale. *Spleen* soft, pulpy. *Kidneys* pale.

2	M.	28	Empyema; drainage tube inserted.	Mar. 10, 1871	Mar. 17, 1871	7 days	Has had two openings made into pleura some months before. Admitted February 20th. Openings enlarged, and a drainage tube put in. *Rigor*, March 10th and 15th; temperature irregular. Highest recorded 103°. Vomiting. Much pain in side. No jaundice. Œdema of one leg. (Notes not perfect.)	Cavity of right pleura almost obliterated. Small cavity containing foul pus. Lungs opposite the wound soaked in pus. *Veins*.—Nothing abnormal noticed. *Lungs*.—Both contain numerous secondary abscesses. *Liver*.—More than a dozen pyæmic abscesses. *Spleen* firm, medium size. *Kidneys* pale and fatty. *Brain*.—Three pyæmic abscesses on surface. *Veins of Leg* plugged from about middle of thigh to iliacs. In femoral and saphenous the clot was broken down into a creamy fluid. Internal iliac vein in same state. No local cause found. No bedsore.
3	M.	2	Fracture of lower jaw (compound), cut lip.	Oct. 15, 1877	Oct. 20, 1877	5 days	No history of any previous ill-health. Fat and healthy. Admitted October 10th. 15th.—Temperature rose to 104° from 100°. 17th.—Some pustules were noticed on the arm, and a patch of brawny cellulitis on forearm. 18th.—Swelling increased; general œdema of arm. Became semi-comatose, and died on the 20th. No diarrhœa or vomiting. No rigors or convulsions. Temperature irregular; highest 105·5°. *Blood* examined on the 18th; nothing abnormal.	*Wound* on face foul and sloughy. *Veins* of face healthy. *Glands* below jaw inflamed. *Lungs*.—One small infarct in left lung. *Brain* congested, a little lymph on surface of pons. *Other organs* perfectly healthy. *Arm*.—Brawny swelling; serous infiltration; no pus. Median vein full of grumous broken-down clot. Axillary closed by a firm thrombus. Chains of granules with independent movement found in serum from arm and in broken-down clot. Post mortem twenty-five hours after death. Body kept in ice.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
4	M.	45	Tumour of tongue (found afterwards to be a gumma) removed by ecraseur.	Feb. 8, 1875 ?	Feb. 24, 1875	About 16 days.	Patient in good health. Urine normal. Operation February 8th. 12th.—Temp. 103·6°; wound sloughy. 17th. Temperature still high; sloughs have separated. Much swelling in glands of neck. 18th.—Red patch over left inner condyle of humerus. 19th.—Phlebitis of internal saphenous, which is varicose; swelling of feet; temp. 99·8°; no rigors. 20th.—Temp. 105·5°; vomiting. 21st.—Extension of the inflammation in arm. 22nd.—The same. 23rd.—Three or four ecchymoses on right hand. 24th.—Phlebitis extending; temp. 102·6°. He died July 24th.	*Stump of tongue* sloughy. Pus in glands about sterno-mastoid. *The red patch* on the left leg, on being cut into, showed diffuse suppuration in the sub-cutaneous tissue. Similar condition in forearm. *Veins* of forearm healthy. Veins of leg filled with clot, not softened. *No petechiæ. Heart* large and fatty. *Lungs* congested. *Peritoneum* containing a few ounces of reddish fluid. *Liver* 87 oz., soft. *Spleen* large and pulpy. *Kidneys* cloudy, swelling.
5	M.	52	Stricture of urethra; external urethotomy.	Sept. 3, 1876	Sept. 14, 1876	11 days	Admitted September 3rd. Rigor same day. Perinæal section on 4th. Large perinæal abscess found. Temperature fell, and patient at first improved. On the 11th rigors returned with vomiting and hiccough. On the 12th two rigors. 13th.—Swelling and pain in left forearm and right calf. Temperature during rigors 106°; irregular.	Incision into membraneous part of urethra. *Wound* sloughy. *Lungs* congested. *Spleen* a mere pulp. *Liver* and *kidneys* swollen. No secondary abscesses. Recent ante-mortem clot in right posterior tibial veins.

6	F.	44	Scirrhus of right mamma; abscess in axilla.	Feb. 11, 1878	Feb. 17, 1878	Cancerous tumour of right breast, adherent to skin. Right axilla full of offensive pus. Small collection of pus on dorsal surface of lower end of metacarpal bone of right little finger. Joint beneath contains pus. Left short saphenous *vein* full of puriform fluid. Both sterno-clavicular *joints*, both shoulders, and right knee full of purulent fluid. *Pericardium*, 1 oz. of dark brown fluid. *Lungs* much congested; bloody fluid in bronchi. *Heart* flabby and decomposed. Great post-mortem staining. *Liver* full of secondary cancer. *Spleen* 7 oz., pale, and very friable. *Kidneys* swollen. Intestines distended with gas. (Post-mortem about twenty-four hours after death.)

TABLE V.—PYÆMIA. GROUP III.: CASES IN WHICH THROMBOSIS AND SOFTENING OF THE CLOT OCCURRED, WITHOUT THE FORMATION OF SECONDARY ABSCESSES.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	M.	32	Lumbar abscess -	Jan. 12, 1878	Jan. 15, 1878	3 days	Admitted in November 1877, with a large fluctuating swelling on right side of lumbar spine, and a deep sore to the outer side of this. January 8th.—1½ pint of fœtid pus evacuated. 12th.—Rigor; temp. 105·9°. 14th.—Temp. 104·8°. 15th.—Died.	Great emaciation. One or two *petechiæ* on lower limbs. *Large psoas and iliac abscess* reaching to upper part of thigh, full of foul pus; hip-joint opened by sloughing in front; caries of third upper lumbar vertebra. *Lumbar glands* swollen. A few *sub-pleural petechiæ*. *Lungs*, marked hypostatic congestion. *Spleen* 6 ins. in vertical diameter, soft, and swollen. *Kidneys* acutely swollen. *Femoral vein*. — Firm adherent decolorised thrombus from one inch below Poupart's ligament for three inches. Below this the clot is softened into a creamy fluid.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
2	M.	37	Primary amputation in lower third of thigh.	July 31, 1875	Aug. 4, 1875	About 4 days.	Admitted July 29th, 1875. Diffuse inflammation spreading from stump; purple discoloration of flaps; foul discharge. No rigors; some vomiting; no marked diarrhœa; foul tongue; marked dyspnœa from the day after the operation. Steady high temperature, about 103° to 104°. Died 6 days after the operation; jaundiced tint of skin three days before death.	*Decomposition* far advanced 24 hours after death. Beyond congestion of the *lungs* there was no other noteworthy pathological appearance in the viscera. The *veins* of the stump were swollen, and contained clots extending some distance upwards. These were not decolorised, but were friable and loosely adherent.
3	F.	50	Excision of varicose veins; dressing at first antiseptic, but this was discontinued.	Feb. 13, 1875	Feb. 18, 1875	5 days	Admitted February 6th, 1875. A diffused blush of redness round the wounds, extending for some distance; surfaces of wound unhealthy; discharges decomposing. Some chilliness, but no actual rigors. Temperature not very high, about 101° to 102°; two sudden rises to 105°. Tongue dry and foul; no marked diarrhœa or vomiting; dyspnœa. Resp. 42 days before death; muscular tremors and clonic spasm of left arm.	The *veins* in both legs above the points operated on contained unhealthy clots, but higher still they were filled with firm adherent coagula; there was much blood-staining of endocardium. *Blood* coagulated as usual. *Lungs* congested at lower lobes. *Spleen* large and pale, but not particularly soft. *Liver* healthy. *Kidneys* small and granular. Post mortem 11 hours after death.
4	F.	48	Abortion; retention of part of placenta.	Sept. 23, 1876	Oct. 10, 1876	Uterus enlarged; inky-coloured patch seen outside at posterior and upper part; inside, corresponding to this, was a shreddy mass of black colour, apparently a retained piece of placenta, stained by perchloride of iron. *Veins.*—Left ovarian contained clot reaching nearly to vena cava; at highest part firm, pale, and non-adherent; lower down firm and adherent; lower still puriform and diffluent at a spot at which a vein from the uterus joined it. *Lungs.*—Congestion and œdema at posterior parts. *Heart* pale and flabby. *Liver* contained bubbles of gas of decomposition; pale and flabby. *Spleen* large and soft; bubbles of gas. *Kidneys* pale, decomposed; no marked staining of intima of vessels; no petechiæ anywhere. Post mortem 18 hours after death.	

TABLE VI.—PYÆMIA. GROUP IV.: CASES IN WHICH THROMBOSIS AND SOFTENING OF THE CLOT OCCURRED WITHOUT THE FORMATION OF SECONDARY ABSCESSES IN THE LUNG, BUT WITH SECONDARY INFLAMMATIONS ELSEWHERE.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	F.	52	Ulcer of the leg; amputation.	June 30, 1877	July 9, 1877	9 days	Very foul old ulcer. Amputation below knee. June 23rd.—Much blood lost; temperature rose on third day after operation to 101·5°; fell on fifth to normal; rose again by evening of sixth to 103°; fell to 99° on seventh. On the eighth day there was a *chill*; temp. 101°. On the eleventh day, temp. reached 104°. It remained high, with variation of about two degrees till seventeenth day, when she died. Before death it rose to 106°. No more rigors; pains in joints. Excess of white corpuscles in blood. No bacteria.	*Wound.*—Nearly closed; only small sinuses left. *Vein.*—In upper part of superficial femoral for about 1½ inch a pale thrombus, firm, adherent. Below, it was softened into pinkish, puriform fluid. Firmer in Hunter's canal. Below this the vein was empty, and its walls intensely inflamed. Inner coat stripping off like a diphtheritic membrane. This condition ceased 1½ inch from the wound; below this the vein was healthy. Foul *abscess*, size of hazel nut, in stump between tibia and fibula. *Heart.*—Walls soft and fatty. *Lungs.*—Hypostatic congestion. *Liver* swollen. *Spleen* rather large, not soft. *Kidneys* swollen. *Joints.*—Both elbows and right knee full of pus.
2	M.	50	Stricture of urethra; forcible dilatation.	Oct. 3	Oct. 13, 1871	10 days	Stricture some years. Admitted on September 7th.—Forcible dilatation shortly after. On October 2nd, an abscess was noticed to be forming in the perineum. Temp. high, but not above 103·2°. One rigor. 9th.—Temp. for last three days about 102°. Very dry tongue, vomiting. Pains all over body and in right knee joint. Albumen in urine.	Fistula and abscess cavity in perineum. Pelvic cellulitis; pus infiltrating cellular tissue. Large abscess beneath gluteus, communicating with pelvis by great sciatic notch. Foul abscess at root of penis. *Dorsal vein* lost in the abscess. Prostatic plexus of veins full of puriform fluid. No *visceral* abscesses. Numerous small abscesses in *rectus abdominis*. Pus in right *sterno-clavicular articulation*. *Spleen* small and firm. *Kidneys* pale. *Lungs*, hypostatic congestion.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
3	M.	42	Pen-holder in bladder; cystotomy.	Nov. 5, 1874	Nov. 16, 1874	11 days	Admitted October 30th, and operated on at once.—Rigor, November 5th, not repeated. A diffuse blush, spreading from the wound to the buttocks, appeared on the 4th. Began to complain of joints on the 7th. Profuse perspirations at first; skin dry at end of case. Profuse diarrhœa. Temp. about 103°, for last week, moderately steady. Icteric tint of skin before death.	Median lithotomy wound, surface sloughy. *Veins* dissected out, a large decolorised clot not breaking down was found in part of the prostatic plexus. *Bladder* inflamed. No pyelitis. *Joints.*—Left elbow, left knee, right knee full of pus. *Muscles.*—Scattered patches of suppuration in vasti of left thigh, reaching as high as the neck of the femur. The pus had no connection with the veins of the muscle. Pus in the sheaths of the extensor tendons of left arm. *Kidneys* swollen. All *other organs* healthy. No post-mortem staining. No petechiæ.
4	M.	44	Stricture of urethra	Mar. 5, 1875	Mar. 11, 1875	6 days	Admitted February 11th, for stricture.—Repeated attempt to pass instruments. 12th.—He had a rigor after the passage of a No. $\frac{1}{3}$ silver. March 6th.—Complained of pain over pubes; rigor; sudden rise of temp. to 104·5°; high temp. maintained. Complained of some pain in various joints. Became delirious. Red blush on back of wrist; effusion into joints; same in left wrist.	Some false passages. Abscess lying between the triangular ligament and the neck of the bladder. The *prostatic plexus* of veins was implicated in the abscess. Internal iliac contained a softening thrombus. *No visceral abscess. Spleen*, 13 oz., pulpy. *Liver and Kidneys* swollen and pale. Joints not examined after death.

5	M.	24	Pericæcal abscess -	Sept. 22, 1877	Oct. 11, 1877	19 days	Illness commenced September 21st, with pain, referred to epigastrium. 22nd.—Violent rigor and vomiting. Admitted September 25th.—Great pain and tenderness in right iliac fossa; tongue foul; spleen can be felt enlarged. 26th.—A rigor. 27th.—A rigor; vomiting; epigastric pain. 28th.—Three rigors. 29th.—Great tenderness in epigastrium. The symptoms remained much the same till death. Repeated rigors; vomiting; tenderness and pain in abdomen. Distension of abdomen, and ten days before death slight jaundice. Temp. extremely irregular; highest 106°. October 2nd. During last three days fell to 102, and remained tolerably regular.	Slight icteric tint. Early decomposition. Diffuse suppuration in subperitoneal tissue behind cæcum. Acute diffuse peritonitis, apparently starting from this point. *Cæcum* healthy. Small concretion in vermiform appendix; not impacted. No perforation. Some excess of fluid in pleuræ and pericardium. No petechiæ. *Lungs* congested. *Liver* very large. *Portal vein* contained creamy pink fluid, and about 1½ inches from liver an adherent yellow thrombus. Walls inflamed. Purulent fluid in branches in liver. Liver studded with points of suppuration. *Spleen* 7 oz., soft and greasy. *Kidneys* slightly swollen. *Other organs* healthy. Marked post-mortem staining from early decomposition.
				Date of Admission.	Date of Death.			
6	F.	57	Cancer of liver; gall-stone ulcerating into portal vein; phlebitis.	Feb. 7, 1876	Feb. 18, 1876	Emaciation. Slight icteric tint. Recent peritonitis, with localised collections of pus about liver. *Portal vein* opened by ulceration into a cavity containing pus. Branches of vein intensely inflamed, and containing softening clot. Several softening patches in *liver* substance, like early "pyæmic abscesses." *Spleen* soft, several recent infarcts. *Kidneys*.—One or two recent embolic patches. *Lungs* hypostatic congestion. *Heart*, recent endocarditis, vegetations on valves of left side.		
7		57	Secondary amputation of thigh.	Feb. 17, 1876 (Amputation Feb. 28).	Apr. 10, 1876	Emaciated. *Amputation wound* almost healed, only a few sinuses remaining. End of bone bare. Two small abscesses, apparently perfectly circumscribed, were found in the medulla at the lower end. *Veins*.—Femoral above junction of saphenous, and circumflex veins full of "softened puriform clot." This extended into common iliac. Saphenous and circumflex veins filled with similar clot. *Lungs* congested, otherwise normal. *Liver* two or three small softening infarcts in right lobe. *Spleen* very soft and friable. Numerous softening infarcts. *Kidneys*.—Cortex pale.		

No.	Sex.	Age.	Primary Disease.	Date of Admission.	Date of Death.	Post-mortem Appearances.
8	F.	21	Tumor of palm of hand; secondary growths in axilla; foul bed-sore.	Feb. 22, 1876	May 11, 1876	Tumor in hand had not been operated on. On the axilla was a sloughy mass of glands. *Pleuræ.*—A little recent lymph in left pleura. *Lungs.*—Grey hepatisation of lower lobe of left lung. No infarcts. *Pericardium.*—A very little recent lymph. *Liver* fatty. *Spleen* normal size, firm. *Kidneys* pale. *Veins.*—Common iliacs, internal and external iliacs, upper part of femorals and lower end of vena cava filled with adherent decolorised clots softening in the centre to a puriform fluid. Both shoulder *joints* and right elbow disorganised, and filled with offensive pus. Body greatly emaciated.
9	M.	36	Amputation of thigh for suppuration in knee-joint.	Nov. 16, 1877	Nov. 27, 1877	Wound granulating, flaps deeply undermined. Lower end of shaft of femur bare. Medulla healthy. No general cellulitis of stump. Sheaths of femoral vessels, thickened and infiltrated, but free from pus. Superficial femoral vein firmly plugged by healthy clot at its divided end, above this as far as the first inch of the external iliac vein, the vein is swollen and filled with softened clot, and above this again a solid healthy plug. Internal saphenous and profunda veins also contain partly softened clot as far as the valves, next to the main trunk, beyond this as far as traced empty and healthy. Glands in groin slightly swollen. *Lungs.*—Marked hypostatic congestion, some broncho-pneumonia. *Liver* large and soft. *Spleen* 8 oz., enlarged, but moderately firm, readily breaking down. *Kidneys* large and pale. *Joints.*—Pus in right shoulder.

TABLE VII.—GROUP V. PYÆMIA, IN WHICH THROMBOSIS AND SOFTENING OF THE CLOT OCCURRED WITH ABSCESSES IN THE LUNGS AND IN OTHER VISCERA AND TISSUES.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	F.	60	Compound fracture of radius and ulna.	Mar. 23, 1872	Mar. 30, 1872	7 days	An habitual drunkard; bloated. Admitted February 28th, 1872. Had broken the arm the day before. Pus formed and burrowed in all directions. Constitutional symptoms not grave at	*Stump* unhealed and sloughy. *Veins* deep and superficial, filled with breaking down clots as high as shoulder; no osteo-myelitis. Subserous petechiæ on heart and lungs. Marked

							first. Temperature little above normal. By March 19th the discharge was very foul; sloughing. Temp. 100°. Dry tongue. Very ill. March 22nd.—Amputation. After the operation there was no improvement. Tem. about 103·5°. Tongue dry; drowsy; muttering delirium. Finally diarrhœa and death, March 30th.—No rigors at any time.	post-mortem staining. One secondary abscess in lower lobe of left *lung*. Numerous abscesses in the liver. *Spleen* and *kidneys normal*.
	2	M.	7	Disease of hip-joint. Acute necrosis after operation.	Oct. 6, 1873	Oct. 27, 1873	11 days	Admitted May 27th. Various incisions and gouging operations from time to time. Erysipelas in July. On October 6th the child's temperature rose, and the lips of the wound swelled. 7th.—Whole thigh swelled; temp. 105°; delirium. On 10th, an exploration was made. The whole shaft of the femur was found to be necrosed; periosteum loose everywhere. *Rigor* (the only one) followed this exploration. From this time to death there was a high irregular temperature; delirium; sweet smell of breath; convulsions. 12th.—Discharge very profuse. Progressive loss of strength. 20th.—Jaundice. 27th.—Death.	Whole shaft of femur bare; lower epiphysis loose. Knee-joint opened and full of pus. Necrosis quite recent; no new bone under periosteum. *Veins*, one inch from vena cava; a firm red clot. Thin decolorised clot for a little distance. At groin and in upper part of thigh, veins contain puriform fluid. *Lungs*.—Numerous abscesses in both lungs. *Brain*.—Abscess, size of nut in left temporo-sphenoidal lobe. *Other organs* healthy; no petechiæ. No post-mortem staining.
	3	M.	18	Wound of knee. Secondary amputation.	July 25, 1878	Aug. 6, 1878	12 days	Admitted June 24th. Attempts made to save joint; first by antiseptic treatment; this failed; then by laying the joint freely open on July 8th. Amputation in lower third of thigh, 24th. Boy very weak and emaciated at time. Had had prolonged high fever. On 25th, a rigor which was not repeated.	Greatly emaciated. *Wound*.—No union, foul, sloughy. *Veins* plugged as high as groin. Puriform fluid in common femoral, in centre of adherent decolorised clot. Veins from bone quite healthy. *Bone*.—Lower inch necrosed; medulla gangrenous for some distance; no general osteo-myelitis. *Lungs*.—

No.	Sex.		Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
							Temperature remained high and somewhat variable, highest 105° on July 30th. Profuse sweating at times without rigor. No vomiting or diarrhœa. Discharge abundant and offensive from wound. Aug. 2nd, became delirious. No other special symptoms.	Pneumonia of whole lower lobe of left lung; breaking down into cavities containing pus; right healthy. *Spleen* small and firm, numerous infarcts just commencing to soften. Other organs healthy.
4	F.	36	Gangrenous inflammation of face.	Jan. 9 (?)1878	Jan. 16, 1878	About a week.	Admitted January 4th. Enormous swelling of lower lip and cheek; scattered points of suppuration. No cause known. It commenced December 26th as a pimple. Incisions were made, but the brawny inflammation continued to spread. No rigors at any time. Strength failed. Tongue became foul and dry. No vomiting or diarrhœa. Delirium three days before death. January 15th.—Left facial paralysis. 16th.—Semi-comatose. Temperature steady; highest, 12th, 104·2°. After 13th gradually fell to 97° before death. Glands below jaw swollen during whole time.	Skin of cheek and forehead undermined by pus and shreddy sloughs. Right cornea had sloughed. *Veins* were traced into the gangrenous mass, and as far as traced were healthy. The *glands* could not be examined. *Heart.*—Large ante-mortem clot; no petechiæ. *Lungs.* — Numerous infarcts in both lungs. *Liver* 63 oz., soft, dark coloured. *Spleen* not particularly soft. *Kidneys* marked cloudy swelling. *Blood.* — Coagulated; no post-mortem staining. *Joint.*—All healthy. *Brain.*—Abundant lymph in sub-arachnoid space at base. Small circumscribed abscess size of pea close to Island of Reil.
5	M.	40	Epithelioma of skin of leg. Amputation.	June 22, 1872	June 24, 1872	2 days	Admitted June 11th. Amputation through left knee on the 12th. Arteries very atheromatous, great difficulty in arresting hæmorrhage. For next few days temperature about 101° to 102°. Discharge foul. Severe secondary hæmorrhage on the 16th. 18th. — Again severe hæmorrhage; ligature of femoral; stump very foul.	*Stump* unhealed, very foul; abscesses extending up the thigh from the wound. *Veins.*—Femoral and profunda filled with puriform broken down clot. *Lungs.*—Right, one small abscess, left, riddled with abscesses. *Kidneys.*—Pale and fatty; other organs healthy. *Joint.*—Right knee full of pus.

							Complained of pain in right knee on the 20th. 22nd.—A severe rigor; pains all over body and down right leg; no other marked symptoms.	
6	M.	24	Scalp wound; osteomyelitis of occipital bone.	Aug. 11, 1875	Aug. 16, 1875	5 days	Ten days after the wound he had a severe rigor; not repeated. Was admitted August 11th. Had throbbing pain in head; no paralysis; urine healthy; temp. 104°, steady; had profuse diarrhœa. On the 12th well-marked jaundice; pain in region of liver; bloody expectoration; confusion of intellect; died comatose.	Bare bone on occipital; outer table cut away. Diploë soaked in pus; a little pus between bone and dura mater; yellowish soft clot projecting from small *vein* into longitudinal sinus close to Torcular Herophili. *Brain* healthy. Sub-pericardial *petechiæ*. *Lungs* congested, three abscesses. *Liver* 99 oz.; innumerable abscesses not limited to surface; pus offensive, but did not blacken lead paper. *Spleen* large and soft. *Kidneys* swollen.
7	M.	31	Compound fracture of leg (bedsore). Primary amputation of right leg.	Jan. 19, 1871 or Mar. 1.	Mar. 20, 1871	? 20	Patient was admitted January 16th, 1871, and primary amputation below the knee performed. Three days after, the 19th, he had a rigor. Rigors were repeated frequently till February 11th, then there was a long interval till March 1st; during this time the patient remained very weak, but had no symptoms of any "blood poisoning." On the 1st the veins of opposite leg were found to be plugged. A large *bedsore* had formed. After the rigors were repeated he gradually died (notes not perfect). The patient seems to have recovered from the first attack due to the stump, and died from a second arising from the bedsore.	Great *emaciation*. *Petechiæ* on front of chest. On side of amputation *veins* are filled with breaking down clots right up to the vena cava and down to entrance of profunda, thence to the stump they are healthy; veins in left side are also diseased. Left common iliac obliterated for one inch, its walls being in apposition and firmly glued together. *Veins* leading from bedsore to right iliac full of broken-down clot. *Lungs*.—Left, six infarcts, not softened; right, five or six similar infarcts. *Liver* healthy. *Spleen* large and soft, two abscesses. *Kidneys* swollen, one small abscess. The source of infection was evidently the bedsore. The stump was healthy.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
8	F.	34	Small bony tumour removed from zygoma.	Feb. 27, 1877	Mar. 3, 1877	4 days	Small bony tumour in front of right ear, six years growth. Admitted February 7th, 1877. Tumour removed on the 15th; profuse hæmorrhage; great difficulty in securing vessel. Did very well till the 27th; wound almost healed; suddenly seized with dyspnœa; loss of power in legs. 28th.—Complete paralysis of left side; temp. 102·2°. March 1st.—Red swelling on back of right wrist; temp. 103°. 3rd.—Partially comatose; no rigors noted.	*Wound* apparently healthy. *Veins.*—Softening clot in facial vein extending into jugular. *Heart* 14 oz., in inter-ventricular septum a small abscess by side of a vessel. Aortic valves "seat of ulcerative disease;" from the apex of two of them depended one clot as large as an almond. The endocardium was ulcerated over an area as large as a sixpence. *Lungs.*—Right, incipient pneumonia of lower lobe; left œdematous; three "pyæmic abscesses." *Liver* soft. *Spleen* 14 oz., one secondary abscess. *Kidneys* 12 oz., yellow infarct in left. *Brain.*—Purulent arachnitis right side; right middle cerebral plugged at division; tissue round softened; small abscess over right corpus striatum.
9	M.	39	Compound fracture of right tibia and fibula. Secondary amputation. Simple fracture of left leg and sternum.	Oct. 6, 1877	Oct. 13, 1877	7 days	Admitted September 22nd. Profuse suppuration of fracture; rapid loss of strength. Bedsores on the 30th. October 6th.—Three rigors; temp. 105°. 7th.—Amputation at knee. 8th.—Rigor. 9th.—Rigor; "wound fairly healthy." 11th.—Discharge very offensive; wound not so well. 12th.—Rigor; tongue dry and brown. Died on the 13th.	*Wound.*—No union; very foul. *Veins.*—Femoral vein in Hunter's canal plugged, and small vessels in neighbourhood of vein filled with pus. *Inguinal glands* on right side enlarged. In cellular tissue of right iliac fossa, and in that surrounding the psoas muscles as high as the kidney, and in psoas itself, recent extravasations of blood. *Joints.*—Right knee full of pus. *Left saphenous vein* full of pus. Fracture of left tibia into knee-joint. *Sternum.*—Sloughy condition of fascia

				Date of Admission.	Date of Death.	
						between pectoralis major and minor; "mousy" odour. Comminuted fracture of sternum bathed in dirty pus. *Heart.* — Petechiæ on left auricle. *Lungs.* — Recent lymph in pleura; sub-pleural petechiæ; small patch of "lobular pneumonia" coated with lymph; central suppuration. *Liver* 97¾ oz., wedge-shaped masses of congestion. *Spleen* soft. *Kidneys* apparently healthy.
10	M.	35	Amputation of thigh.	May 6, 1875	June 20, 1875	Abundant breaking-down clot in veins of stump. Both *lungs* contain numerous softening infarcts and "abscesses"; pleura over these inflamed. *Liver* pale and soft. *Spleen* 7½ oz., large, soft, numerous softening infarcts.
11	F.	33	Syphilitic ulceration of rectum.	April 1, 1875	June 14, 1875	Great emaciation. Extensive ulceration of rectum, sinuses burrowing in all directions. *Veins* of pelvis filled with puriform fluid. *Pleura.*—Some purulent fluid. *Lungs.*—Many pyæmic infarcts, numerous sub-pleural ecchymoses. In right lung the infarcts are softened, and the surface covered with lymph. *Heart.*—Muscular substance soft and mottled, no peri- or endocarditis. *Peritonitis* over spleen. *Spleen* large and soft, numerous infarcts, some softening. *Liver* large and friable. *Kidneys* large and pale.
12	M.	33	Inflammation of corpus spongiosum. Phlebitis, stricture of urethra.	Jan. 26, 1876	Feb. 3, 1876	Great inflammatory swelling of corpus spongiosum; no suppuration. One of the *veins* about the neck of the bladder full of softening clots, some like grumous pus. *Blood* for the most part fluid, dirty purple colour. *Lungs* congested; in right lung one large and several smaller infarcts. *Pericardium.*—One ounce turbid serum, recent lymph. Surface of *heart* mottled with yellowish softening patches and spots of intense injection. *Liver* soft, friable. *Spleen* 10½ oz., soft. *Kidneys* soft and flabby, a few scattered points of intense redness, with yellow softening centre.
13	M.	46	Perinæal abscess and stricture; incisions.	Dec. 26, 1874	Jan. 17, 1875	Body much decomposed in 24 hours. Numerous incisions in perineum. *Veins* about neck of bladder contained semi-purulent coagula. *Lungs.*—Infarcts and "one or two abscesses." *Spleen* soft, 7 oz. *Liver* fatty. Recent suppuration on back of hand and about ankle. *Joints* not affected.

TABLE VIII.—PYÆMIA. GROUP VI. CASES IN WHICH THROMBOSIS AND SOFTENING OF THE CLOT OCCURRED WITH SECONDARY ABSCESSES ONLY IN THE LUNG.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	M.	54	Necrosis of femur -	Oct. 6, 1877	Oct. 12, 1877	6 days	Disease of lower end of left femur for thirty-five years. Twenty years ago some necrosed bone was removed. Sinus never healed. Lately discharge and pain increased. Admitted September 17th, 1877. October 3rd.—Operation under chloroform. 5th.—Discharges very foul. 6th.—Temp. rose to 103·8°. 7th.—Rigor, temp. 105·8°. 9th.—Swelling of left calf. —10th.—Two rigors. Irregular temp., 99·2° to 103·2°. Pyæmic odour. Tongue dry. 12th.—Died.	Left leg much swollen. No petechiæ. Glands in groin swollen. *Veins.*—Superficial femoral healthy till entrance of veins from bone. Then filled with softening creamy clot. *Lungs.*—Numerous pyæmic infarcts softening. None in left. *Heart.*—Subpericardial petechiæ. *Spleen* large, not soft. *Kidneys* swollen.
2	M.	18	Amputation of thigh for disease of knee.	June 12, 1877	June 20, 1877	8 days	Amputation of thigh for disease of knee, May 26th. Severe hæmorrhage on 27th. From the effects of this he gradually recovered, and the stump began to heal. On June 12th he had a severe rigor, and from that time till his death the case followed the ordinary course. Repeated rigors at irregular intervals. High temperature; irregular. Gradual emaciation and exhaustion; sweating; delirium.	Amputation of left thigh in lower third. *Stump* swollen. Wound nearly united, only a few sinuses left. End of bone necrosed in lower half inch. Periosteum separated from bone, along whole of shaft, by offensive pus, as high as hip, which is full of pus. Marked osteo-myelitis. *Veins* from bone filled with softening puriform clot; femoral at entrance of profunda contains similar clot; elsewhere healthy. *Glands* in groin swollen. *Lungs.*—Softening infarcts in both lungs. *Heart.*—No subserous petechiæ. *Liver* pale. *Spleen* enlarged but firm. *Kidneys* slightly swollen.

3	M.	43	Epithelioma in glands of groin secondary to penis.	Feb 15 (?), 1871.	Feb. 22, 1871	About one week.	Penis amputated November, 1869. Recurrence in glands. Ulceration November, 1870. Had shivering before admission. Right leg swollen. Deep foul ulcers in groin. Six days in hospital. Seven rigors in first four days. None afterwards. Highest temp. recorded 105°. Temp. irregular. High at death, 104°. No other special symptoms noted.	Foul sloughy excavation in right groin. *Veins.* — Right femoral inflamed; broken down red dish débris of clot. No puriform fluid in vein. "Pyæmic abscesses" in both lungs. Left pleura full of offensive pus. *Liver* healthy. *Spleen* swollen and soft. *Kidneys* congested.
4	M.	16	Necrosis of tibia. Amputation of thigh.	After Dec. 9	Dec. 26, 1871	?	Admitted for necrosis of tibia, October 13th, 1871. Amputation December 2nd. Discharge offensive after first few days. Did well at first. Afterwards high temperature and rigors. Temp. record lost. Notes imperfect.	Emaciated. Skin yellowish, with purple patches. *Stump* swollen. Skin edges of flaps united except opposite bone. Abscess burrowing along bone to small trochanter. Osteo-myelitis and necrosis. Medullary canal putrid. *Veins.*—Profunda full of puriform fluid. Inflamed. Femoral after entrance of the profunda in the same state. *Lungs.* — Numerous softened infarcts. *Other organs* free from infarcts. Albuminoid degeneration. Numerous subserous *petechiæ*.
5	M.	36	Conical stump. Removal of bone from femur.	Dec. 19, 1870	Jan. 1, 1871	12 days	Amputation August, 1870. Admitted into hospital November 25th, for unhealed conical stump. December 15. —Stump reopened and two inches of bone cut off. 19th.—A rigor; repeated the same day, and none after that. Temperature high; not very irregular. Osteo-myelitis followed the operation. Pus was let out of the medullary canal by removing some prominent granulations. Notes not perfect.	Stump open and unhealed. Bone bare and necrosed; periosteum separated as high as the hip-joint; pus in hip-joint. *Veins.* — Puriform fluid in veins leading from the necrosed bone. Numerous secondary abscesses in *lungs*. *Liver* pale and fatty. Other organs healthy.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
6	M.	20	Conical stump. Re-amputation through the knee.	Dec. 25, 1870	Dec. 30, 1870	5 days	Amputation at knee by long anterior flap. December 22nd.—Intermediary hæmorrhage; flap sloughed on third day. Pus formed in the synovial pouches of knee, and was pent up in them; offensive smell. First rigor 25th. On 28th was delirious. 29th.—Pleurisy diagnosed. 30th he died. Temp. high (104°), irregular.	*Stump* sloughy and unhealed. Foul abscess burrowing up thigh. *Veins.*—Femoral opened on face of stump unclosed. It contained yellow pus-like fluid, containing leucocytes and innumerable granules. This extended to two inches below entrance of profunda, where was a soft, slightly adherent clot. Walls of veins inflamed. *Lungs.*—Numerous pyæmic abscesses in both lower lobes. Pint of yellow fluid in left pleura.
7	M.	36	Compound fracture of femur. Fissures into knee-joint.	Feb. 1871	Feb. 23, 1871	12 days	Small wound closed with collodion. Admitted January 27th. The wound did not close; there was free suppuration. Temp. rose to 103° on February 11th, with a rigor. Steady high temp., 103° to 104°, till 16th; rigor on 16th; three rigors on 17th; one rigor on 20th. Died on 23rd. Thigh much swollen; tenderness in left groin. Delirium; smell of breath, &c.	Wound open. Pus burrowing amongst the muscles of the thigh. *Veins.*—Popliteal, femoral, and iliacs to vena cava plugged; in the iliacs the clots were recent and non-adherent; lower down adherent and softening; small veins of thigh filled with puriform fluid; coats of veins inflamed. *Bone* healthy. *Knee-joint* opened by the fracture; full of pus. Numerous pyæmic abscesses in both lungs. *Liver* healthy. *Kidneys* swollen. *Spleen* mottled, not unnaturally soft.
8	M.	39	Compound fracture of fibula.	Feb. 3, 1871	Mar. 7, 1871	32 days	Admitted January 12th. Wound three inches long on outer side of leg. Fracture of fibula; went on well till February 3rd; slight shiver. Rigors 4th, 5th, 6th. 7th (2), 16th. Temperature not very high; 102·8°	Large abscess between superficial and deep muscles. Pus very offensive. *Bone* ununited; no osteo-myelitis. No necrosis. *Veins.*—Femoral immediately below entrance of profunda plugged with firm adherent clot. In

							highest recorded. No antiseptic dressing to wound; discharge profuse; some burrowing of pus. Pneumonia was diagnosed on 16th; pleurisy on 11th. Delirium with progressive loss of flesh and strength were the only symptoms noted. Temperature fell before death to normal.	the centre this was softening into puriform fluid. Below, the veins were filled with puriform fluid; this fluid contained numerous leucocytes and granular matter. *Lungs.*—Numerous abscesses in both lungs. Many had burst into pleura. Offensive pus in both pleuræ. Post-mortem twenty-four hours after death. Weather cold.
9	M.	37	Ununited fracture of tibia. Ends of bone excised.	Feb. 9, 1874	Feb. 18, 1874	9 days	Admitted January 27th, 1874. Resection of bone February 4th. Erysipelas appeared on 9th, and continued to spread till death. No rigors. Temperature record lost. The erysipelas was apparently simple cutaneous.	*Wound* sloughy. *Veins.*—External iliac and femoral as low as profunda filled with adherent, firm, decolorised clot; profunda in same state. Below this in places the clot had broken down into a puriform fluid. *Bones.* Osteo-myelitis of upper fragment. *Lungs.*—Numerous secondary abscesses. *Liver* pale. *Spleen* large, soft, pale. *Kidneys* swollen and pale.
10	M.	9	Disease of hip-joint. Excision.	Nov. 20, 1874 (?)	Nov. 26, 1874	6 days	Admitted 24th April, 1874. Deep abscess over hip-joint; aspirated three times; then opened; prolonged suppuration. Temperature above normal, usually about 100·5°. November 19.—Excision of hip. After the operation the temperature rose to 104°. During the following days the temperature was irregular, but no rigors were noticed.	*Wound* sloughy and foul. Abscesses burrowing in various directions. *Veins.*—Inflamed from external iliac to knee. The clot in parts breaking down into puriform fluid. This condition extends into the profunda. *Bone.*—The whole shaft of the femur is bare of periosteum; medulla gangrenous. *Lungs.*—In middle of upper lobe of right lung is a "pyæmic abscess." In the lower lobe of the left lung is an area of "diffused pneumonia." *Liver* albuminoid. *Spleen* pale and firm. *Kidneys* pale.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
11	F.	36	Strumous arthritis of elbow; amputation.	April 5, 1876	April 8, 1876	3 days	Admitted March 29th; inflammation of elbow; patient much emaciated. On April 2nd a free incision was made and much pus let out; patient very ill. Amputation on April 3rd; no rigors; profuse sweating; irregular temperature, highest 104°. Gradually sank and died April 8th.	*Stump.*—No union of flaps; basilic vein up to axilla full of green pus; brachial veins healthy. *Lungs.*—Numerous pyæmic abscesses in both lungs. *Liver* pale. *Spleen* large, soft, and swollen. *Kidneys* swollen. Other organs healthy.
12	M.	38	Compound fracture of skull; wound of lateral sinus.	Aug. 19, 1876	Sept. 15, 1876	26 days	Admitted August 16th; compound depressed fracture of skull; wound of lateral sinus; depressed fragment removed and sinus plugged; plugs became foul. Violent rigor on 19th, repeated on 20th; plugs removed, bleeding returned; plug replaced. No rigor till 24th. From this time till death there were repeated rigors —39 in all—sometimes three a day; he had also over 100 epileptiform convulsions, chiefly during the early part of the case; no marked symptoms beyond the rigors and gradually increasing emaciation and weakness; temp. very irregular, highest 107°, lowest 95°.	*Wound* sloughy; dura mater commencing to slugh opposite wound; no general meningitis; large foul abscess in neck. *Veins.*—Lateral sinus full of débris of a putrid, broken-down clot; the plugs had been put actually into the sinus; the phlebitis and suppuration had extended through the mastoid foramen and caused the foul abscess in the neck; jugular healthy. *Lungs.*—Numerous pyæmic abscesses, varying in size from from pin's head to filbert, chiefly in lower lobes; slight pleurisy. *Liver* healthy. *Spleen* soft and swollen. *Kidneys* swollen.
13	M.	49	Old buboes - -	Nov. 3, 1873	Nov. 8, 1873	5 days	Chancre three months ago. Admitted for buboes September 2nd. 3rd.—Laid freely open. 10th.—Incision on right side; apparently doing well. Attacked by erysipelas 30th; left very weak after the erysipelas disappeared; abscesses burrowed towards	The abscesses had burrowed into pelvis and towards hip-joint. *Veins.*—Large softening clot in internal iliac. *Heart* soft and fatty. *Lungs.*—Pyæmic abscesses in right only. *Liver* fatty. *Spleen* large and firm. Other organs healthy.

							pelvis; wounds foul and sloughy. Rigors November 3 (2), not repeated; temp. 106·5°, irregular. Died November 8th; no special symptoms noted.	
14	M.	7½	Double hip disease; excision of left hip.	?	Dec. 7, 1874	—	Admitted October 16th; double hip disease. 17th—Abscess opened left. 28th.—Excision of left hip; wounds foul; much discharge. November 12th.—Erysipelas; this disappeared. Elevation of temp. 103° November 29th. Bedsores; gradual exhaustion.	Excision surface rough and carious; right joint almost destroyed. *Femoral vein* on right side plugged to ham; on left side profunda full of softened clot, femoral plugged with firm clot. *Lungs.*—Lower lobes on both sides contain numerous softening infarcts. Other organs healthy.
15	M.	18	Exostosis of lower end of femur; suppuration of knee; secondary amputatation of thigh; bedsore.	Feb. 13, 1874	Feb. 19, 1874	6 days	Admitted December 6th, 1873. Exostosis removed on the 10th. Pus found in ham, and somehow got into the knee-joint. Acute destructive inflammation of knee; temperature averaged about 102°, steady; rapid emaciation; discharge abundant. Amputation above knee. February 15th.—Large bedsore. *Rigors.*—On the 13th two, 15th two. Died 19th. No other special symptoms noted.	*Stump.*—Slight superficial union. *Bones* and *veins* healthy. *Foul sloughy bedsore* over sacrum; veins leading from this to internal iliac full of puriform fluid. A softening thrombus projected from internal into common iliac. *Lung.*—Five small softening infarcts in left lung; none in right; some subpleural petechiæ. *Other viscera* healthy.
16	M.	21	Old disease of knee; abscess in thigh; amputation at hip.	Jan. 16, 1870	Jan. 26, 1870	13 days	Admitted September 29th. Knee injured a year before. Extensive suppuration in thigh; abscesses opened. Had erysipelas in November. Amputation at hip January 10th. Vomiting for several days, but rallied from shock of operation. 20th.—Pain in course of iliac vessels; no repair of wound. 24th.—Slight convulsions; rapid respiration; cough. No rigors recorded.	*Wound.*—Little union, surfaces of flaps sloughy in many parts. *Veins.*—Femoral contained "pus," clothing extended up to the common iliac, upper end not breaking down. *Bones.*—Acetabulum rough and eroded. *Lungs.*—Softening infarcts in left lung; none in right. *Liver* fatty. *Spleen* tolerably consistent. *Kidneys* swollen, some small isolated patches of congestion.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
17	M.	45	Gonorrhœa; cystitis; diffuse cellulitis of scrotum; suppuration in left spermatic cord; orchitis.	May 1, 1874	May 19, 1874	18 days	Admitted April 24th. Extensive suppuration in scrotum and along left spermatic cord. Severe rigor May 1st.	*Lungs* congested and œdematous, with pyæmic patches. Abscess in *prostatic plexus of veins*, and in cellular tissue of left iliac region. Grey tubercle in lungs, cheesy matter in left cord.
18	M.	13	Round-celled sarcoma of forearm; amputation of arm.	Feb. 28, 1872	Mar. 9, 1872	9 days	Admitted February 3rd. Amputation on the 8th. Went on well till the 15th. Secondary hæmorrhage; flaps opened up; then went on well till the 28th, when temperature suddenly rose to 104°; no rigor. Temperature remained high and irregular; highest 106·4°, lowest 102°. Tongue dry; sweating freely at intervals, but no rigor till March 8th, not repeated. Pain in chest; friction heard both sides; delirium; emaciation. No diarrhœa; discharge very offensive.	*Wound* unhealed, flaps covered by feeble granulations. *Bones.*—Periosteum completely separated, acute osteo-myelitis, suppuration of medulla; pus in shoulder-joint of affected side. *Veins* leading from bone full of broken-down clot; divided end in stump healthy. *Lungs* full of secondary abscesses. *Spleen* and *kidneys* slightly swollen and congested. Other organs healthy.
19	M.	54	Retention of urine; stricture; puncture of bladder by rectum; external urethrotomy.	June 26, 1877	June 29, 1877	3 days	Admitted June 21st, 1878. No instrument could be passed. Bladder punctured from rectum. Temperature slowly and steadily rose till 25th, when it reached 103°. Tongue white and moist; bowels relaxed. 26th.—External urethrotomy; *rigor* in the evening. 27th.—Temp. 100°; seemed better. 28th—Profuse hæmorrhage from rectum. 29th.—Died.	A foul sloughy cavity was found between the bladder and rectum. The *veins* were involved in the sloughy mass, but could not be dissected out. *Lungs.*—"Secondary abscesses." All other organs healthy.

20	M.	32	Compound fracture of tibia and fibula.	June 4, 1877	June 6, 1877	2 days	Admitted May 16th. Water dressing. Was a healthy man. Delirious till 20th; then did well till June 24th; sudden rise of temp. to 103·4°; 28th.—Wound unhealthy; œdema of limb. 4th.—Rigor, repeated same day. 5th.—Discharge less, then another *rigor*. Amputation through knee. Posterior tibial vein plugged; popliteal the same. Became drowsy; breathing laboured. Died twenty-eight hours after amputation. Temp. at time of death 104°.	Body well nourished. *Wound.*—Flaps ecchymosed. *Veins* plugged up to external iliac. *Glands.*—Swollen in groin. *Heart.*—Slight excess of pericardial fluid; sub-pericardial petechiæ. A few recent firmly adherent vegetations on mitral valve. *Lungs* large branch of left pulmonary artery plugged with recent clot; numerous subpleural extravasations. In lower lobe of left lung a patch of lobular pneumonia, with vessel plugged. Lung generally œdematous, with numerous small arteries plugged with decolorised clot. *Liver.*—Gorged with blood. *Spleen.*—Large, almost diffluent. *Kidneys.*—Large, smooth, soft.
21	F.	3½	Disease of hip-joint; diphtheria; acute osteomyelitis of femur.	Mar. 28, 1871	April 1, 1871	3 days	Admitted January 24th, 1871. Miserable emaciated child. Abscess opened. Continued discharge. (No temperature record.) March 28th.—Child very ill; thick discharge from nose. 29th.—Discharge from nose and ears; sweating; high temperature. 31st.—Large purpuric spots on legs. April 1st.—Died.	Post-mortem 8 hours after death. Decomposition commenced; purpuric spots all over body. *Wound.*—Sinuses about hip. Joint disorganised. Whole periosteum of femur loose; medullary canal full of pus. *Veins.*—Femoral up to the entrance of profunda full of puriform fluid. *Lungs.*—Three or four recent infarcts. *Kidneys.*—Pale and large. *Pharynx, &c.*—Areolar tissue about pharynx œdematous. Mucous membrane of nose, pharynx, larynx, and trachea covered with typical diphtheritic false membrane. *Uterus.*—Surrounded by clot ⅛ths inch thick.

No.	Sex.	Age.	Primary Disease.	Date of Admission.	Date of Death.	Post-mortem Appearances.
29	M.	19	Abscess of internal ear; phlebitis of right lateral sinus and internal jugular vein.	Jan. 15, 1877	Jan. 16, 1877	Dura mater over petrous portion of temporal bone swollen; ashy grey. Small quantity of lymph in cavity of arachnoid. Subarachnoid fluid turbid. *Veins.*—Right lateral sinus from torcular full of puriform fluid. Right jugular vein inflamed, coats thickened; small adherent coagula on surface; no large adherent coagula. *Pleuræ.*—Full of offensive purulent fluid. *Lungs.*—Contain very numerous softening infarcts. *Spleen* 9½ oz., swollen, soft. *Kidneys.*—Swollen cortex.
23	M.	36	Compound fracture, with depression of left parietal bone.	Sept. 17, 1877	Se	Fracture of skull had been trephined. bone around bare and necrosed. Dura mater wounded. "Purulent matter" over whole left hemisphere. Right lateral sinus contains decolorised and puriform clot. Several fractured ribs on left side. *Left pleura* full of offensive pus. *Right lung* gorged with blood. No infarcts. *Left lung* collapsed; numerous pyæmic abscesses. *Spleen* very large and soft. *Kidneys* swollen. *Liver* pale and soft.
24	M.	32	Compound dislocation; secondary amputation through elbow.	March 27	April 17	Wound ununited. Pus burrowing up arm. Bracheal vein normal. In the middle of the arm a branch joining the basilic vein is filled with decolorized and partly softened clot. *Lungs.*—Marked hypostatic congestion. Numerous infarcts in both lungs, commencing to soften; chiefly in lower lobes. Slight pleurisy over the softening infarcts. *Liver* large and fatty. *Spleen* 10 oz.; very large and soft. *Kidneys* swollen.

TABLE IX.—GROUP VII. ACUTE NECROSIS, OR INFECTIVE PERIOSTITIS.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mostem Appearances.
1	M.	16	Acute necrosis of femur.	Aug. 7, 1874	Aug. 12, 1874	5 days	Taken suddenly ill on the 7th. Pain in limbs. Delirious. 9th —Kept bed from onset. Right leg amputated two years before for disease following a blow. Admitted on the 10th. Swelling and redness of right arm from shoulder to wrist; delirious. Temp. 103·9° to 106°. Swelling of left knee and thigh. Rapidly sank and died Aug. 12th.	Post mortem twelve hours after death. Serous fluid in left knee. Serous infiltration of muscles of thigh. Two thirds of length of femur bare, and surrounded by pus. No necrosis in arm. Excess of synovia in elbow and wrist. Veins of limbs healthy. *Pericardium.*—Abundant recent lymph. *Heart.*—Substance inflamed. Abscess commencing to form in one part. *Lungs.*—One small abscess in right lung. *Spleen* 4 oz., soft. *Blood* from heart dark maroon colour. No bacteria to be found in it.
2	M.	16	Acute necrosis of humerus.	Sept. 15, 1877	Sept. 26, 1877	11 days	Sudden invasion on 15th; severe headache. Better on 16th. On 19th herpes appeared on lips, and pain in shoulders. No rigors. On 21st was seen by a doctor, who diagnosed acute rheumatism. Delirious at night. Swelling of arm not noticed till 24th. Arm swollen from elbow to shoulder. Temperature 103°, great prostration; temperature gradually rose to 108° on 26th, shortly before death; no great variations; sweating; no incisions in arm; trace of albumen in urine.	Post mortem eighteen hours after death. No superficial petechiæ. Swelling of *left arm.* Tissues infiltrated with serum. Periosteum separated from upper two thirds of humerus by grumous offensive pus. Shoulder-joint and line of epiphysis healthy. Small subperiosteal abscess close to elbow. Elbow full of pus, not offensive. Medulla free from pus. *Veins.*—Veins of arm as high as axilla and as low as bend of elbow full of puriform clot. *Pleuræ.*—Recent lymph. *Lungs* gorged with

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
								blood, numerous infarcts, mostly hæmorrhagic, a few softening. *Pericardium.*—Numerous petechiæ, recent pericarditis. *Heart.* — Numerous patches of inflammation in muscular substance. One or two small abscesses in wall of left ventricle. *Spleen* 7½ oz., somewhat swollen. One small abscess. *Kidneys* swollen. Two minute points of pus.
9	M.	19	Acute necrosis of ulna and humerus.	Feb. 2, 1878	Feb. 16, 1878	14 days	February 2nd.—Complained of pain in right *wrist*; no injury. Swelling of arm on the 10th. Admitted on the 11th. Pus evacuated from tissues about *elbow*-joint. Temp. 100·2°. Temperature on 16th 104°. No rigors. Notes imperfect.	Two recent incised wounds on outer side of right forearm. Fœtid purulent infiltration of muscles of forearm.—Middle third of ulna entirely deprived of periosteum. Lower two inches of humerus also bare. *Veins* of forearm full of "pus." Veins of arm in similar condition. *Glands* in axilla swollen. *Joints.* — Pus in right elbow and shoulder. *Lungs* slightly œdematous and congested.—*Liver* pale. *Spleen* 5½ oz., natural. *Kidneys* pale. Other viscera normal.
4		16	Acute necrosis of left tibia.	Probably Jan. 10, 1878.	Jan. 16, 1878	6 days	No history of injury or acute specific fever. January 5th. — Felt pain in legs. 7th.—Took to bed; treated for rheumatism; no rigors. Admitted on the 11th. Very ill; acute pain in right side; tongue red; pulse feeble. Left leg swollen; œdematous. Femoral glands enlarged. Saphena vein at	Post mortem five and a half hours after death. Wounds perfectly free from any odour of decomposition. Quite clean. Pus alkaline. Left *saphena vein* full of grumous, broken-down clot, containing no leucocytes. Multitudes of granules and some moving organisms. *Glands* in groin swollen.

							inner side of knee; marked out by superficial redness; plugged with clot. Urine healthy. Free incisions. Whole tibia bare. Pus red. *No organisms* to be detected in it. *Temperature* steady, between 102° and 103°. *Blood examined* on the 13th and 14th. *No organisms.* Red corpuscles form masses and not rouleaux. No vomiting; no diarrhœa; delirium; convulsions. During last twelve hours of life temperature rose from 102° to 105°. Died at 5.30. At 6.30 temperature had risen from 105° to 108·9°.	Left tibia in greater part bare. Lower epiphysis loose. *Heart.*—Numerous subpericardial petechiæ at back of heart. *Lungs.*—Numerous softening infarcts in both lungs. *Liver* slightly swollen and soft. *Spleen* not abnormally soft or large. *Intestines* healthy. *Kidneys.*— Numerous infarcts, some softening. *Blood* from right auricle at post mortem contained moving jointed organisms. *Joints.*—Left knee and ankle and right elbow full of pus.
	5	M.	8	Acute necrosis of tibia.	?	March 4	?	Admitted February 8th. Acute periostitis following slight injury. Pale, thin. Pulse 140. Fluctuation over whole tibia. Effusion in knee. Incision. 11th.—Another incision. 24th.—Amputation refused by parents. March 4th.—He died.	*Bone.*—Great part of tibia bare. Some new bone forming in places beneath the periosteum. *Lung.*—Several secondary abscesses in lung. Foul pus in left pleura. *Kidneys* congested. *Other organs* healthy. Blood fluid twenty hours after death.
	6	F.	15	Acute necrosis of tibia.	?	Jan. 11, 1874	?	Admitted January 2nd. High temperature. Abscess opened. No rigors noted.	*Bone.*—A small extravasation of blood beneath the periosteum. Suppuration in medulla opposite this point. Another patch of inflamed medulla near upper epiphysis. *Lungs.*—Numerous softening, hæmorrhagic infarcts. Other organs healthy.
	7	M.	A boy.	Acute necrosis of femur.	July 3, 1877	July 26, 1877	23 days	July 3rd.—Came home from school ill. Feverish. Pains all over. Left knee red and swollen. 6th.—The same, but delirious. Great pain. Temp. 105°. Admitted into medical ward.	Abscesses on both ring fingers opening the joints. No other joint superficially affected. *Pericardium* distended with puriform fluid. *Pleuræ.*—Both coated with recent lymph. Numerous

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
							9th.—Thin and feeble. Tongue dry. Temp. 102°; pulse 132; resp. 60. Left thigh flexed on pelvis. Great pain. Pericardial friction. *Urine* normal, sp. gr. 1022, high coloured. 10th.—Screaming from pain. 14th.—Diarrhœa to the 18th. Sweating freely. 22nd.—Pleuritic effusion. Much toothache. Some stumps were removed. Some abscesses formed over the knuckles, and were opened. Collapsed, and died on the 26th. Diagnosis not made during life. Supposed to be rheumatism. Temperature about 102° to 103°. No rigors.	sub-pleural extravasations. *Lungs.*—Bronchial tubes filled with airless puriform fluid. *Heart.*—Surface of a curious mottled colour. Vegetations on surface of apex of heart. Some recent fibrinous clots on mitral valve. Substance of wall mottled on section. *Liver* congested. *Spleen* large and firm. *Stomach.*—Rugæ stained with effused blood. *Kidney* peels readily on external surface. Some small white spots, in some cases surrounded by extravasated blood. Similar spots in pyramids. *Head.*—Extravasations of blood under pericranium. *Brain.*—Gelatinous clot in longitudinal sinus. Excess of fluid beneath arachnoid. *Left hip.*—A collection of pus was found separating the periosteum from the bone about the trochanter and upper part of the left femur.
1	M.	9	—	Jan. 13, 1877	Jan. 22, 1877	9 days	Admitted January 15th. For days before complained of pain in legs; no rigors. On admission, pallid and ill-nourished; pains in legs and front of chest; no redness or swelling; extension of legs; increased pain. Urine slightly albuminous. Temp. 103°. Fell to normal on 17th; slightly elevated till 20th, when it reached 103°, and remained high till	Right cheek swollen and red; a small abscess at root of right bicuspid. *Heart.*—Pericardium contains ½ of a pint of thick pus; shaggy lymph. Two ulcerated patches, excavating muscular substance; a small abscess in the muscular wall. *Lungs.*—¼ pint purulent fluid in left pleura; lungs congested. *Liver* congested. *Spleen* normal. *Kidneys.*—Con-

					death. No diarrhœa; no vomiting; increased pericardial dulness; friction; delirium from 17th. Swelling of face and toothache about 15th or 16th. Cyanosed before death.	gestion and scattered points of suppuration in both.
				Date of Admission.	Date of Death.	
9	M.	3	Acute necrosis of left tibia.	July 18, 1876	July 25, 1876	Body much decomposed; large part of tibia necrosed; some new bone formed in it; abscesses opened in leg by recent incisions. Large abscess in left axilla. *Lungs* congested and œdematous. *Pericardium* contained 4 oz. of pus. No abscesses in substance of heart. *Veins* not mentioned.
10	M.	11 mos.	Acute necrosis of femur.	?	Sept. 13, 1876	Shaft of femur completely bare. Abscesses had been opened by free incisions. *Pleura*.—In right side 8 oz. of creamy pus. *Lung* collapsed and congested. Other viscera normal.
11	F.	15	Acute necrosis of tibia and shaft of humerus.	Feb. 12, 1877	Feb. 16, 1877	Greater part of shaft of tibia bare. Incisions had been recently made down to the bone. Epiphyses loose. Medulla pink and grumous. Cancellous tissue presents patches of injection. Pus between periosteum and bone at lower end of humerus. *Basilic vein* full of puriform fluid, extending into axillary vein. Veins of leg healthy. *Heart*.—A few scattered points of commencing myocarditis. *Pericardium* healthy. *Pleuræ*.—Numerous ecchymoses, lymph, no fluid. *Lungs* contain very numerous infarcts. *Liver* pale. *Spleen* $3\frac{1}{2}$ oz., not enlarged, soft, greasy. *Kidney*.—Very few minute infarctions in cortex.
11	F.	13	Acute necrosis of fibula and ungual phalanx of right middle finger.	?	Jan. 11, 1875	An incision over outer side of right fibula, just above malleolus. Lower third of shaft of fibula bare. Ungual phalanx of right middle finger bare. *Pleuræ*.—Recent pleurisy on left side. *Lungs*.—Numerous infarcts in both lungs; none softened. *Heart* healthy. *Liver* soft. *Spleen* large and soft, numerous infarcts, some softened. *Kidney*.—A few small infarcts. *Veins*.—Nothing abnormal found on careful examination.

Table X.—Group VIII. Ulcerative Endocarditis.

No.	Sex.	Age.	Primary Disease.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Examination.
1	F.	35	Ulcerative endocarditis.	—	Feb. 28, 1878	—	Attack of "ague" October, 1877, returned December, 1877. Repeated rigors. Admitted February 12th, 1878. Loud systolic murmur. Gradually got worse. Irregular temperature, but rigors ceased; tongue dry, sordes. Became insensible, and died on the 28th. Albumen in urine. Spleen could not be felt during life. Abdomen swollen and tender.	Body fat. A few recent ecchymoses in subcutaneous fat. *Heart* enlarged. Endocardium of left auricle opaque. Mitral valve presents an irregular mass of vegetations, $\frac{3}{4}$ inch by $\frac{1}{2}$ inch; some sub-endocardial infiltration in valve, ventricular surface of valve irregularly eroded, small adherent clots, some firm, some soft and crumbling. Pericardium contained 1 oz. of turbid fluid, numerous petechiæ. One small embolic abscess in substance of right auricle. *Lungs.*—Here and there small scattered ecchymoses. *Pleura* contained turbid fluid, no lymph. *Peritoneal cavity.*—One pint of turbid, semi-purulent fluid, no lymph. *Liver* acutely swollen. *Spleen* enlarged, moderately firm, not friable, no abscesses or infarcts. *Kidneys* small.
2	M.	22	Ulcerative endocarditis.	—	June 21	—	Two months before admission had pains in loins and forehead. Shortly after he had shiverings every evening. A few days before admission symptoms ceased; very weak. Admitted May 7th. Emaciated, anæmic. Later on he had rigors almost daily. Highest temperature about 104°, very irregular. Vomiting; progressive emaciation. June 18th.—Blood examined,	Slight œdema of ankles. Numerous cutaneous *petechiæ*. *Heart.*—Minute subpericardial petechiæ. On mitral valve two large vegetations, firm but friable; some calcareous granules in parts. Ventricular wall thickened. *Lungs.*—A few minute hæmorrhages beneath visceral pleura; lungs congested. *Liver* dark brown and firm. *Spleen.*—Pulp soft, two large, old in-

							some granular matter, excess of white corpuscles, great deficiency of red. Gradually became weaker and died. Urine albuminous.	farcts (caseous), and several smaller infarcts immediately beneath the capsule. *Kidneys* large, white, no infarcts.

Table XI.—Spontaneous Pyæmia and Septicæmia.

No.	Sex.	Age.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
1	M.	48	Jan. 10	March 19	3 months.	January 10th.—Shivering on coming out of a hot room; cough next day. Since then *frequent rigors*; sweating at night; bowels regular. On January 30th, February 4th, and March 11th nearly got well, but relapsed soon after. Admitted March 14th with above symptoms and pain in back of neck, head, and calves; pulse 114; liver dulness slightly increased; no other marked physical signs. 15th. — Cough less. 16th. — Eruption like impetigo on legs; delirious at night. 17th.—Eruption increased; delirium worse. 18th.—About twenty hard, inflamed, cutaneous lumps on each leg, size of peas; some suppurating, red raised patches, size of crown-piece, on forearm. 19th. — Delirium constant; eruption increased. Blood extravasated in some of the suppurating points; patch on forearm fluctuating; glans penis apparently gangrenous. Died. Patient was a butler.	*Teeth.*—Right molar decayed, bone healthy; no inflammation. *Skin.*—Bullæ on face, legs, and arms; purple, with red areola. Several lumps due to small abscesses in the muscles of forearm. Glans penis swollen; dark purple red. *Joints* perfectly healthy. *Larynx.*—Ulceration of membrane lining the ala of the thyroid cartilage, surrounded by congestion and ecchymosis. The patch was made up of numerous minute purulent heads. *Lungs.*—Both intensely congested; numerous collections of minute abscesses were found, with distinct pyæmic characters; two or three larger accumulations of unhealthy pus, size of filbert. *Heart.* — Nothing abnormal. *Liver*, 72 oz. *Kidneys* congested. *Spleen* much congested, granular on surface. *Glans penis* infiltrated with pus.

No.	Sex.	Age.	Date of Invasion.	Date of Death.	Duration of Case.	Symptoms.	Post-mortem Appearances.
2	F.	21	Nov. 12, 1876	Nov. 24, 1876	12 days	Good previous health. November 12th.—Began to suffer from headache and cough, with slight expectoration. 17th.—Languid pain in chest, general aching. 19th.—Vomited. 22nd.—Admitted. A rigor; bowels regular. (On 19th was out of doors, but was very ill in the evening; temp. 105°). 23rd.—Two loose stools, dark colour; twelve distinct rose spots; constant delirium; temp., M., 102·8°; E., 104°; loud systolic murmur. 24th.—Vesicular eruption on fingers and toes and forehead; in some places pustular; temp. 104·8°; died; delirious till death.	*Skin.*—Purple hæmorrhagic spots at ends of fingers and toes; a few vesicles on front of legs, some becoming pustular; herpetic spots on scalp and forehead. *Subcutaneous tissue.*—Slight ecchymosis under scalp, corresponding to the herpetic spots. *Brain.*—Small collection of pus under visceral arachnoid; effusion of blood into left hemisphere and in cerebellum. *Heart.*—Beneath endocardium, chiefly at upper part of right ventricle, were a few small spots, the size of pins' heads, apparently minute collections of pus; zone of congestion round them. *Lungs* congested; in upper lobe two or three cone-shaped congested spots. *Liver.*—Two or three spots, mottled with yellow, beneath the capsule. *Spleen* normal. *Intestine.*—Spots of minute suppurations in mucous membrane; nothing like typhoid ulceration. *Kidneys.*—Specks of pus in cortex, mostly immediately beneath capsule.
3	F.	39	Sept. 15 ?	Oct. 14	29 days	Patient, a nurse, admitted Sept. 21st. Weak for some months; sore throat and pains in neck; fauces and tonsils red and swollen; no rash. In six days throat got well, but pain still continued in neck; patches of erythema appeared on both sides of neck, behind parotids; margins raised; pain and swelling of left hand and shoulder day before admission. 21st.—Pain, redness, swelling of both hands; pain in left shoulder; pain in left face and deafness of left ear; teeth on that side carious. 27th.—Better; swelling of joints gone; bright	*Heart* 12 oz., soft and flabby. *Lungs* congested at bases. *Liver* 64 oz., pale and fatty. *Spleen* 4 oz., soft. *Intestines* congested; hæmorrhagic patches in mucous membrane. *Kidneys* slightly congested, otherwise normal. *Blood* very fluid 36 hours after death.

						rose rash, itching; blotches ¼ in. diameter, not raised, disappeared on pressure. October 4th. — Rheumatic pains gone; red papulæ rash; rigor; temp. 104·5°; pulse 132; throat sore again; offensive breath; purulent ophthalmia. 8th. — Lips swollen; sordes; rash darker, resembling purpura, but disappearing on pressure; pulse 140; 10th.— Vomiting; blebs on skin; eyes very bad. 12th.—Diarrhœa; no vomiting; rash nearly gone; epidermis desquamating. 14th.— Died.	
4	M.	48	Feb. 28	April 5	36 days	Cough 3 months. February 28th.—Severe rigors. Admitted March 3rd. Jaundiced; liver dulness increased; temp. 103·8°; great abdominal pain; purgatives; jaundice diminished; much cough. He remained unchanged some time, jaundice then increased, emaciation. Four days before death sordes, profuse sweating; red flush on hands and wrists.	*Lungs.*—Right contained at base a small circumscribed area of "secondary pneumonia;" congestion of mucous membrane. *Liver* large, lobules distinctly marked out. *Joints.*—Pus in left sterno-clavicular and wrist joints.

MARCUS BECK.

APPENDIX III.

Notes of Microscopical Examination of Blood during Life.

Case 1. *Acute necrosis of humerus (periostitis, involving elbow-joint); rigors; amputation; recovery.*—George E—, æt. 21. Admitted January 24th. Operation February 1st, 12.30 a.m.

Blood examined February 1st, 3 p.m.—Temp. 101°; pulse 96, full, slightly dicrotous. (Day very cold and ward cold.) Blood coagulates with great difficulty; red corpuscles natural, forming rouleaux slowly. Granular masses of rather large size, consisting of distinct granules, which appear ovoid or round, and measure 1·5 μ in diameter. The granules are of pretty uniform size, much larger than those seen in the white blood-corpuscles. On subsequently squeezing the slide, in order to break up the white corpuscles, the rouleaux separate. Some of the white were squeezed out, but the granular masses thus produced were much less coarsely granular than those above mentioned. Most of the white blood-corpuscles natural, but some only half the diameter of the majority. Proportion of white to red, average 1 to 50. No bacteria or vibriones. (Number of white blood-corpuscles excessive; red normal; granular masses abnormal.)

Temperatures:

January 24th.—E. 101·2°.
25th.—M. 99·8°; E. 104·2; 3 p.m. 102·9°.
26th.—M. 102·4°; E. 103·2°.
27th.—M. 102·7°; E. 103°.
28th.—M. 102·8°; E. 103·8°.
29th.—M.103·7°; E. 104·6°.
30th.—M. 103·4°; E. 101; 3 p.m. 106·8°, shivering.
31st.—M. 98·2°; E. 99·6°.

February 4th, 6.30 p.m.—*Blood* (with Dr. H. U. Smith and Mr. Pitts). White blood-corpuscles decidedly more numerous than before, red scantier; white appear natural; amœboid movements active. Rather less granular matter, but of the same character as before. No bacteria. Temperature is higher again.

6th, 3 p.m.—Temp. 101°; pulse 92, somewhat dicrotous; respirations 28. Feels better. *Blood.*—Fewer granular masses, but still of the same kind. *White* corpuscles natural; amœboid movements natural; average number in thirty-two fields only 5. No bacteria or micrococci.

Case 2.—Thomas H. H—, æt. 36. Punctured wound of elbow-joint. Fracture of inner condyle of humerus. Rigor February 4th, in morning.

Blood.—February 25th, 5 p.m.—Temp. 103°. Very few red corpuscles; white in very great excess; in a considerable number of fields the average is 1 white to 18 red. Characters of white natural. Very numerous moving particles, some extremely minute, some double-jointed, others in strings, and yet others rod-shaped, with independent locomotive movements, evidently rod-shaped bacteria; these latter measured about 1·5 μ in length, the others too small to measure alone, but their relative size nearly that given in drawings (Plate I, figs. *a*, *b*). Some of the more minute dotted filaments only just visible (with Hartnack, obj. 10, oc. 2).

26th, 6 p.m.—Blood of somewhat dark colour. Red corpuscles natural. White in great excess. A considerable number of small ovoid granules. Some bacteria still seen, dumb-bell shaped, in very active movement, continuing after the fibrin filaments had formed. None of the others seen to-day.

Patient died on March 16th, at 3.15 p.m., examined March 18th, 2 p.m.

Abscess in temporo-sphenoidal and occipital lobes of right hemisphere of brain. Fracture into condyle. Excision of left elbow-joint.

Right lung, 1 lb. 9½ oz.; left lung, 1 lb. 5½ oz.; heart, 13 oz.; liver, 4 lbs. 8 oz.; spleen, 12½ oz.; kidney, 12 oz.; brain, 3 lbs. 1½ oz.

Body of an emaciated male. Rigor mortis well marked in lower extremities. Much lividity of posterior surface of trunk. Slightly jaundiced colour of skin. Left hand and forearm œdematous. Excision of left elbow by vertical incision over olecranon; surface of wound unhealthy. Lower ends of bones in wound-surface necrosed. Veins of left arm healthy. A few old adhesions between outer surface of left lung and chest-wall, also between base of lung and diaphragm. Right pleura healthy. Pericardium adherent over entire surface of heart; old calcareous plate in pericardium, size of a penny, situated in the fibrous pericardium, adjacent to left auricular appendix. Right auricle and right ventricle empty; left auricle empty; left ventricle ditto. Surface of heart coated with fat. Walls of heart pale and flabby. Much post-mortem staining of valves and great vessels. Spleen large, substance rather soft and friable. Both kidneys somewhat pale, otherwise healthy.

Lungs.—Left surface rather emphysematous. Bronchi exude muco-purulent-looking fluid; upper and back part œdematous, otherwise healthy. Right anterior part of upper lobe; surface emphysematous. At posterior part of upper lobe is a cavity, size of pigeon's egg, immediately under pleura, which is adherent to chest-wall. Cavity contains cheesy-looking material. Lower lobe congested, otherwise healthy.

Liver.—Upper surface of right lobe adherent to diaphragm. Gall-bladder contains normal quantity of bile; substance pale and fatty.

Brain.—Veins of pia mater much congested. In interpeduncular space superficial to pia mater is a collection of sero-purulent fluid lying on pons and extending a certain distance down medulla. This fluid exudes from a large abscess cavity situated in right temporo-sphenoidal and occipital lobes, also extending into descending horn of lateral ventricle of same side. Cavity extends also into interpeduncular space beneath pia mater cavity; has tolerably thick walls, and contains a quantity of purulent fluid.

Case 3.* *Amputation of left arm; osteo-myelitis; symptoms of pyæmia; secondary amputation; death.*—W. T.—, æt. 65, watchman.

November 14th, 5 p.m.—Three rigors this afternoon. *Blood.*—Red corpuscles of natural appearance; coagulation natural, but a tendency to rather rapid crenation. White blood-corpuscles in some excess; amœboid movements natural. Numerous minute ovoid granules with separate movements. Rod-shaped bacteria, measuring 1·5 μ to ·3 μ in length, with free independent movements—a number seen.

15th, 6th p.m.—Rigor in morning. Temp. now 97·5°. Delirious; talking much, loudly. Pulse 120, full, somewhat dicrotous. *Blood.*—Red blood-corpuscles scanty but normal, forming rouleaux, though less readily than natural, and tailing off easily, as in anæmic blood. White in great excess, numerous clumps, and also uniformly scattered over the field. Red and white carefully counted in six average fields, proportion 1 white to 25 red; a number of others less accurately counted gave similar results. White blood-corpuscles with very rapid amœboid movements, rapidly becoming granular masses. Very few other granular masses. No free granules. Only one bacteroid body discovered on prolonged search; this about 1·5 μ in diameter, and one highly refractile ovoid body, not freely moving, about ·8 μ.

16th, 7 p.m.—Temp. 100·4°. White blood-corpuscles much more numerous than before, varying a good deal in size. Red not abnormal. Except a small number of minute free granules, nothing seen at first; but after half an hour two or three very minute, almost invisible.

Post-mortem examination (six hours after death).—Weather cold, almost frosty. Body brought down soon after death. Rigor mortis well marked. No petechiæ; no anasarca.

Blood from superficial vein in left groin swarmed with micrococci and bacteria. From another adjacent none at all, and from one in right groin none at all. From a small superficial vein a little below left ankle none at all to be discovered. Blood from cephalic vein of left arm, no bacteria; from left subclavian vein, a few dumb-bells. From right auricle only two bacteria to be discovered. Blood quite natural in other characters; red blood-corpuscles normal.

* Appendix II., Table I., Case 4.

Fluid from right pleura no bacteria; from pericardium no bacteria.
Fluid from pleura and pericardium contained no bacteria.
Discharge from wound swarmed with bacteria.
Joints.—Both knee-joints, ankles, hips, and right shoulder and elbow examined and found to be healthy.
Head, brain, &c.—Dura mater natural. Old softening of the greater part of the right tempora-sphenoidal lobe, and of a large part of the convolutions of hemisphere adjacent, also of nearly the whole of right corpus striatum. Extreme atheroma of cerebral arteries. Nothing else abnormal.
Spinal cord not examined.
Larynx, trachea, and thyroid gland natural.
Pleuræ.—Left, general old slight adhesions. Right normal.
Lungs.—Left somewhat emphysematous; right more so. Some patches of broncho-pneumonia, especially at the upper part of lower lobes of both lungs, much more marked there than elsewhere, especially in left. *No infarcts* whatever to be discovered in any part of the lungs.
Heart.—A very large, loose clot in right auricle; also in right ventricle. No staining of endocardium. Some narrowing both of aortic and mitral orifices (old endocarditis).
Liver.—Soft, pale, greasy.
Spleen.—Swollen, pale, soft, greasy.
Kidneys.—Granular contracted; not very advanced.
Supra-renal bodies natural.
Stomach and intestines natural.
The stump was carefully examined, and nothing of an abnormal character found; the axillary vein showed no sign of phlebitis or softening clot in any part of its course; the axillary artery contained a normal clot.
Note.—This case is an important one. It was clinically a most typical case of pyæmia; the arm was amputated for a very severe burn; osteo-myelitis followed, with symptoms of pyæmia; amputation was performed higher up, but the symptoms continued, and the patient died with all the features of pyæmia; yet after death the most careful examination failed to discover any secondary deposits, or even any secondary lesions such as usually occur. Bacteria were found in the blood in considerable number on only one occasion, two days before death; none at other times.

Case 4. Mary M'G—, æt. 34. Admitted August 8th, 1877. Excision of right knee, August 18th. Amputation, February 13th, 1878. Rigor, February 17th, 3.50 to 4.15 p.m.
February 19th, 5 p.m.—Temp. 102.5°; pulse 120, small and rapid. Anæmic, tongue dry red, somewhat brown in centre, tremulous. Blood from finger has characters of anæmic blood; forms rouleaux naturally, but readily broken up; red scanty, white in slight excess, but nothing else notable in characters. Very few granules; nothing else abnormal.
21st, 4.30 p.m.—Temp. 103°. Blood from finger pale. Red blood-corpuscles natural, but very scanty, white in large number, of pretty natural appearance and equal size; nine fields accurately counted; average number of white in field 16, proportion to red 1 to 50. Some microcytes measuring only 3·6 μ in diameter, of reddish colour. No bacteria; very few granules.
22nd.—Had a slight rigor at 1 p.m. Temp. at 5 a.m. 105°, at 3 p.m. 101°; pulse 108 (moved into small ward). *Blood* now thin and watery, much more so than before. Red blood-corpuscles natural, form rouleaux normally; white to red as 1 to 40. Elongated rod-shaped bacteria now seen, measuring 3·5 μ to 4 μ long; some ovoid bodies with distinct movements, and other jointed ones of smaller size, all with distinct movements. The bacteria move very freely, continuing in movement after the fibrin filaments have appeared. Some ovoid bodies measuring nearly 3·u in length have apparently amœboid movements. *Pus* from wound quite natural; no bacteria or granules.
23rd. 3.30 p.m.—Temp. 105·2° Had a slight rigor at 4 a.m. lasting 10 minutes, temp. 100·4°. Another very slight at 12, temp. 99°. At 3 p.m. temp. 105·2°; injection of carbolic acid given; 3.30, temp. 105·2°; 4 p.m. 103°; pulse 136. small and weak; face flushed, frequent retching and slight vomiting. *Blood.*—Red corpuscles, scanty, form normal rouleaux, easily separated. White blood-corpuscles appear to be much less numerous in proportion than before;

very active in their movements. Proportion 1 to 140 (several careful estimates). Very numerous granules of various size, also very numerous, extremely minute jointed filaments, some only just perceptible, others larger, some in clusters (fig. 1*a*,. Plate I). The average length of the individual granules, as estimated from the length of the chains, was about ·15 μ to ·25 μ. Some larger filaments, nearly ·3 μ long, made up of these. A few distinct bacteria-like rods, from 2·5 μ to ·3 μ long, in no respect differing from ordinary bacteria. The careful examination of several different slides gave precisely the same result.

25th, 6 p.m.—Temp. 102° (very ill yesterday, no rigor. Temp. to-day not over 102°). *Blood.*—Red blood-corpuscles look more numerous, coagula normal. White to red 1 to 45, on average of about fifteen fields; the variation in different fields very slight. There are now very few granular masses, no free granules and no moving particles; no bacteria or micrococci visible on most careful examination.

26th, 5 p.m.—Had a severe rigor this afternoon, beginning at 3·30 and lasting an hour. Temp. at 12, 102·2°; 3.30, 105·2°: 3.45, 106·5; at 4, 104·4°; 5.15, 100·6°; 5.45, 102°. The two last taken during examination of blood.

Blood bright-coloured red. Red blood-corpuscles not so scanty, but of anæmic character, tail out easily but form normal rouleaux. White in much less excess, though still abundant, varying from 7 to 10 in field. A considerable number of fields counted, but the white adhere so rapidly to the glass that the estimate was considered of no value. Proportion estimated about 1 to 100. Very few free granules only after a prolonged search. But both before and after coagulation the following bodies were seen and some of them watched for some time after. (The whole observation occupied an hour, different specimens of blood being examined in succession.)

1. A few ovoid granules about ·5 μ in diameter, with apparently locomotive movements.

2. A few ordinary rod-shaped bacteria, some of elongated ovoid shape, measuring rather less than 1·5 μ in length, with very distinct locomotive movement, lasting after the subsidence of all other movements.

3. Some much smaller bodies, at first rather difficult to see owing to their extreme tenuity; first noticed by their movements, which were of very active locomotive character. They consisted of rows of dots 2, 3, 4 or more in number, forming chains which measured usually not more than ·75 to ·1 μ. Their movements were very active and lasted long after quiescence of the fluid and of Brownian movements. See fig. 1*a*, Pl. I.

27th, 2.15 p.m.—Temp. 99°; pulse 108, weak, not dicrotous. Great pallor; profuse perspiration. *Blood.*—Red corpuscles of natural size and appearance, form rouleaux naturally. White natural, amœboid movements active. Proportion of white to red (average of twelve observations, of which each gave almost exactly the same proportion), 1 to 50. Only one bacterium found, this of a character of two dots, not more than ·4 μ long. One or two other moving granules, but a prolonged search showed no other bacteroid bodies. *Pus* taken from deep-seated granulations contains normal pus-corpuscles and very numerous bacteria, precisely resembling those seen yesterday in the blood, extremely minute, in active movement, and some other free granules of the same kind. There are also one or two larger rod-shaped bacteria. Perspiration from neck, which is very profuse, contains some bodies which are smaller than these rod-shaped bacteria, but larger than the rows of dots seen above.

28th, 5.15 to 5·50 p.m.—Temp. 99·5°; pulse 120. No rigor to-day; temperature was 102° at 11.25 a.m., the highest to-day, 101·2° at 12 midnight, and 99·2° at 2 a.m. Is very prostrate, quite sensible, profuse perspiration, very typical cowshed odour. *Blood* natural; coagulation natural. White corpuscles natural in character; amœboid movements normal; size natural. In about twenty fields, average 11. Only one dumb-bell seen. No bacteria. *Pus.*—Much fewer bacteria than yesterday, and in less active movement; characters otherwise unaltered. Died March 2nd at 8 p.m. Post-mortem 4th, at 3 p.m.

Body of medium-sized female, somewhat emaciated. Stum of amputation through middle third of right thigh. Surfaces of flaps have scarcely any appearance of granulating. Superficial necrosis of exposed end of bone for one inch. Right external iliac vein contains breaking-down puriform clot, which extends into common iliac. A large bedsore over the sacrum commu-

nicates with a large abscess in the cavity of the pelvis on right side, which extends also downwards into gluteal region; the lower part of sacrum and the coccyx are denuded of periosteum and roughened.

Pleuræ.—Normal; *lungs* emphysematous; œdema of lower lobe of right. Right 22 oz. left 15½ oz.

Heart.—Flabby, otherwise natural. Weight 10 oz.

Liver.—45½ oz., flabby, fatty looking.

Spleen.—4 oz. Very soft and friable.

Kidneys.—*Right* shows typical tuberculous pyelitis, ureter and base of bladder also involved. Left natural.

Brain.—Natural.

No secondary deposit or inflammation could be anywhere discovered; the abscess in pelvis was in evident connection with the bedsore over sacrum.

Case 5. *Abscess of groin of doubtful origin; opened by puncture; rigor.*—W. H.—, æt. 45. Admitted January 21st, 1878.

February 4th, 6.30 p.m.—Temp. 102·8°; pulse 108, full, dicrotous. Profuse perspiration. *Blood.*—Enormous increase of white corpuscles—the majority of usual size, but some considerably smaller, and a few distinctly nucleated. In some fields (of No. 10 obj., 2 oc. Hartnack) as many as sixty or seventy, but time failed to count many fields. Red natural but scanty. Many spherical or ovoid highly refractile granules, not very uniform in size, but the majority not more than about ·0015 mm. No moving bacteria.

6th, 3 p.m.—Temp. not taken; pulse 108, somewhat weak and dicrotous. Prostrate. Pain in abdomen. *Blood.*—White corpuscles still in great excess. average in field 30, proportion to red 1 to 24 (twelve observations). White corpuscles not abnormal, only one or two of small size. Very distinct masses of granules (as in Case 1), and when a mass of the same size as a leucocyte was compared with it, side by side, the contrast between the two was very great. The individual granules are ovoid or angular, about ·0015 mm. No bacteria or moving particles. Red corpuscles natural, somewhat scanty.

8th, 3.30 p.m.—Temp. 102·5°. 4 p.m.—Temp. 103·4°; pulse 160. *Blood,* 4 p.m.—Enormous increase of white corpuscles, which average 30 to 40 in field of normal characters. Very numerous granular masses as before; the granules are sharply defined, ovoid, or rounded, some much more definitely so than those seen before. In some cases they are joined end to end. They measure about 1·5 μ in length, and are about ½ or ⅓ of that in width. No distinct moving bodies seen. No bacteria. A few microcytes.

Urine contains a trace of albumen, red blood-corpuscles, and some few pus-cells.

Case 6. *Myositis and cellulitis.* (Case 2, in examination of organs Appendix IV.)—Blood examined by Mr. Wagstaffe, who found very numerous bacteria, mostly of apparently dumb-bell shape.

Case 7. *Rupture of perinæum; operation; pyæmia.*—Amelia S—, female, 35. Blood examined October 24th, at 5 p.m. Temp. 102°. Patient apparently moribund, respiration catching, short restless cough; pulse small, rapid, fluttering. Marked yellowish tint of skin; pyæmic odour and aspect well marked. Red corpuscles appear natural, form rouleaux readily; no irregularity in shape or size. White very numerous, 20 to 35 in field (estimated proportion to red 1 to 20); characters quite natural; none with more than one nucleus seen; amœboid movements active. Masses of granular matter abundant. No bacteria or other moving bodies observed. No free granules. Died October 25th. No post-mortem allowed.

Case 8. *Amputation of thigh; pyæmia.**—Joseph F—, æt. 18, labourer. Thigh amputated May 26th.

June 14th, 1878.—White corpuscles not obviously increased; amœboid movements natural; red corpuscles scanty, form rouleaux fairly well. No bacteria or moving particles.

* Appendix II., Table VIII., Group IV., Case 2.

15th.—No excess of white blood-corpuscles, if anything somewhat scanty, a few of them about two-thirds diameter of the rest. Some bacteria and vibriones in very active movement.

CASE 9. *Fracture of jaw; pyæmia.*—J.C—, June 14th, 1878. Enormous increase of white-corpuscles, estimated 1 white to 20 red. Amœboid movements hardly perceptible. No bacteria.

CASE 10. *Double amputation of legs; pyæmia.*—August 14th, white blood-corpuscles in great excess (10 to 14 in several fields); red normal. No increase of protoplasmic masses. No trace of bacteria or vibriones on prolonged examination.

CASE 11. *Chronic pyæmia, with pustular eruption.* (Case 4, in Examination of Organs, Appendix IV.)—July 11th, 5 p.m. Blood thin and watery. Red corpuscles aggregate with difficulty, but otherwise not notable. White somewhat abundant. Amœboid movements natural, some few nuclei; only one to two minute moving specks. No rod-shaped bacteria. *Pus* from forehead: pus corpuscles ill defined; many vibriones, but no rod-shaped bacteria.
No bacteria were found on two other examinations.

CASE 12. J.W.—Great increase in white blood-corpuscles and granular matter. No moving bodies.

CASE 13. *Amputation of thigh.*—A. F—, male, æt. 17. Examination July 13th. Temp. 103·5°, pulse 100, full, moderately firm, regular; skin dry, hot. Discharge, which has been profuse, began to lessen last night, when rigor occurred. *Blood.*—White 1 to 50 (seven estimations) red, diameter ·0075 to ·0080.
16th.—Considerable increase in white blood-corpuscles, estimated on several observations as 4 white to 100 red. Red form rouleaux, though not easily. No excess of granular matter, no bacteria.

CASE 14. W.H.—August 4th. Great increase in white blood-corpuscles, red natural; no bacteria.
15th.—In twenty fields quite as many white as red corpuscles. White somewhat variable in size, but otherwise natural. A number of motionless, highly refractile granules seen in all the twenty fields examined, and several elongated bacteria seen in all.

CASE 15. Elizabeth H—, æt. 48. Sarcoma of thigh; excised March 26th. Symptoms of blood poisoning 24 hours after operation. March 31st, 9 p.m. A rigor occurred at 7.13 p.m., lasting twenty minutes. Temperature now 102·5°. Patient semi-delirious; profuse perspiration, hurried breathing, pyæmic aspect. Blood of quite normal character. White not obviously increased. Examination, lasting half an hour, showed nothing but two bacterium termo of ordinary character. Died. No post-mortem allowed.

CASE 16. *Amputation of thigh; rigors; pyæmia.*—March 31st. No bacteria. White blood-corpuscules much increased in number.

CASE 17. *Periostitis of tibia.*—Mary A—, æt. 10.
Abstract of History.—Fell and struck her leg on January 2nd, 1879. Swelling and redness on Jan. 4th. Admitted on Jan. 6th. Immediately after admission on Jan. 6th an incision was made, and a quantity of fœtid pus evacuated.
8th.—Spots observed on trunk and limbs about mid-day. No rigors: no diarrhœa. Physical signs of pericarditis. Albuminuria. Tenderness over spleen. Enlargement of liver.
9th.—Blood examined at 8.40 p.m. Enormous increase of leucocytes. Bright, highly refracting granules, about 1·5 μ in masses, seen in great abundance. Smaller bodies, not more than ·4 μ in diameter, in active movement. Rod-shaped and dumb-bell bacteria. Minute, rapidly moving bacteria of much smaller size, similar to those seen in Case 4. Other larger protoplasmic masses of peculiar shape, moving freely and giving out amœboid processes. Died at 9.10 p.m.

Temperatures :—

January 6th.—E, 104·5°.

7th.—7 a.m., 104°; p.m., 103°.

8th.—2 a.m., 104°; 11 a.m., 103·6°; 2 p.m., 103°; 5 p.m., 102·8°; 8.30 p.m., 102·6°; 11 p.m., 103·4°.

9th.—1 a.m., 102·2°; 5 a.m., 102°; 8 a.m., 101·2°; 12 noon, 102·1°; 2 p.m., 103·3°; 5 p.m., 102°; 7 p.m., 102·2°; 8.30 p.m., 102°.

Case 18.* *Ulcerative endocarditis.*—Annie J—, æt. 35. Admitted February 12th, 1878. Died March 1st. Post-mortem eight hours after death. Symptoms of ague, rigors recurring pretty regularly twice a day, between 10 and 12 a.m. and p.m. Temp. 105° or so, falling at times to 94°.

Blood.—February 26th, 5 p.m.—Temp. 97°; pulse 88. Very great increase of white blood-corpuscles—in many fields 20 to 30; red very scanty—in many fields not more than 3 red to 1 white. Red show typical characters of anæmia. White, amœboid movements natural; some few are smaller than natural, others rather larger and distinctly vesicular in the centre, the majority natural. No bacteria, only very few granules, and nothing else abnormal. No other examination of the blood made.

Post-mortem, March 1st (7 hours after death).—Temp. in interior of liver 95·5° F. Body still warm. Abundant sub-cutaneous fat. One or two small ecchymoses in subcutaneous tissue of left leg. Very slight anasarca of feet.

Peritoneal cavity contains about six ounces of semi-purulent fluid. Serous membrane nowhere injected.

Pleura.—Right contains half a pint of turbid fluid, containing flakes of lymph. Left a like quantity, more distinctly purulent.

Pericardium also 1½ oz. of yellowish serum, with some flakes of lymph. Some minute pin's head white spots surrounded by injection over left auricle.

Heart.—Large; both ventricles somewhat dilated, especially right, which contains very voluminous coagula. Pulmonary valve competent. Left ventricle contains a large mass of clot, some of which is of ante-mortem origin. Mitral valve appears to close almost completely, but on the posterior segment is a large mass of fungous vegetations, which projects downwards ¼ inch into the ventricle. Other smaller vegetations on valve.

Lungs.—Congested; hypostatic pneumonia.

Liver.—Enlarged, soft; outline of lobules fat.

Spleen.—Enlarged, firm; no visible infarcts.

Kidneys.—Small, pale, firm. Old and recent infarcts in cortex.

Nothing important in other organs.

Case 19.† *Pyæmia. Lateral lithotomy.* Appendix II. Group I. (*a*), Case 13.—F. B—, æt. 21. Abscesses in the subcutaneous tissue, and one irregular-shaped, softening patch, dirty yellow colour in the spleen.

Blood.—Examined one day before death for bacteria, with a purely negative result.

Case 20. *Pyæmia; Lateral lithotomy; wound sloughy.* Group I. (*a*), Case 14.—W. C—, æt. 18. This patient was so irritable and nervous that the blood could not be examined during life. At the *post-mortem* examination, seventeen hours and a half after death (weather cold), a drop of fluid blood obtained from a superficial vein showed a great excess of white corpuscles. These were excessively granular but of good size. The red corpuscles were with very few exceptions crenated. They did not form rouleaux, but were heaped up in irregular masses in which the outlines of the individual corpuscles could not be recognised. No rod-shaped bacteria or micrococci were seen, and nothing presenting independent movement. The pus from the pleura showed pus-cells of ordinary shape and size, but excessively granular. The liquor puris was teeming with bacteria; some rod-shaped, some pointed, none showed any very entire movements.

Case 21. *Pyæmia.* Appendix II. Group I. (*a*), Case 5.—In this case the blood was examined repeatedly during life. As the patient became gradually

* Appendix II., Table X., Group VIII., Case 1.

† Cases 19, 20, and 21 are furnished by Mr. Beck.

weaker the blood became paler in colour and poorer in red corpuscles. There was slight actual, and very great relative increase of the red corpuscles. Some small granules were observed at various times, but nothing was seen that could be said with any certainty to be an organism.

Note on Microscopic Examination of Blood and Organs with reference to Bacteria.

There is no reason to believe that the micrococci found in these cases were different from those found by other observers in decomposing pus. There may be a doubt whether the larger bodies, viz. those measuring 1·5 μ or more, are spores of bacteria or larger micrococci. Again, the rod-shaped bacteria from 1·5 to 3 μ in length differed in no distinguishable respect from ordinary bacterium termo. The bodies concerning which the greatest doubt exists are the very minute rods seen in these cases, and more particularly observed in the blood and wound secretions of Case 4.

It seems more than probable that these bacteria are identical with those observed by Dr. Burdon Sanderson and others in the peritoneal fluid of animals suffering from artificial septicæmia. Dr. Sanderson's description is as follows:—"The peritoneal fluid was viscid, and coagulated imperfectly or " not at all. It contained pus-corpuscles, which were relatively not numerous; " the plasma (liquor puris) often exhibited a tremulous movement, which " under high powers is found to be due to the presence of minute rods. With " lower powers particles cannot be distinguished; all that can be seen is the " peculiar tremulous movement of the liquid, which, however, is sufficiently " characteristic. In addition to this appearance, the liquid contains other " actively moving rod-like bodies, larger than the particles above mentioned, " but still not greater than $\frac{1}{100}$ of a millimètre (2 μ) in length." "In the most rapid cases dumb-bells and chains are mostly absent." And further on—"In all the acutely infected animals to which the table relates, in which " the blood was examined, it exhibited microscopic appearances which were " characteristic and unequivocal. They were of two kinds. The blood " plasma exhibited more or less distinctly tremulous movement of the particles " above described, and the colourless corpuscles often contained rods in their " substance, and that the blood discs seemed to adhere to each other, the " blood possessing a remarkable viscidity."* This description, with which I was not acquainted at the time, might have served almost equally well for the description of the blood of McG—.

The description given by Koch of the bacilli which he found in the blood of animals inoculated with decomposing blood may also be advantageously compared. "If the animals died after *inoculation* with decomposing or " septicæmic blood, then only the small bacilli appeared in the blood, and " that without any exception, and always in large numbers. These bacilli, " which lay either scattered or in small clusters between the red blood-" corpuscles, measured from ·8 to 1 μ. Their thickness, which could only be " judged of, not measured, is about ·1 to ·2 μ. The bacilli in septicæmic " blood are often seen two connected together, either in a straight line or " forming an obtuse angle. Longer chains to as many as four bacilli also " occur, but rarely. Without staining, the bacilli are remarkably difficult to " recognise in fresh blood, even when one already knows their form, and I " have not been able to arrive at any certainty as to whether they possess an " independent movement. Their relation to the white blood-corpuscles is " remarkable. They crowd in them and increase in number in them. One " often finds hardly a single white blood-corpuscle in which there are not " bacilli to be seen."

Dr. Koch also states that he has found that these bacilli grow like splenic fever bacilli, but not like these to long threads, but only to thick heaps consisting of separate bacilli. In some cases also he has seen spores appear in the bacilli.

The results of these two observers accord with my own observations on the bacteria found in acute septicæmia in the guinea-pig. The blood when first examined, and also the serous exudation which, as observed by Dr. Sanderson,

* "Rep. of Med. Off. Privy Council and Loc. Govt. Board," New Series, No. VI., p. 63.

is remarkably viscid, swarmed with these very minute rods, which are very difficult to distinguish unless by staining, or until the eye is accustomed to their minute difference in refraction from the fluid in which they float. By cultivation of these in serum or aqueous humour, there were obtained, in successive generations, first spores, then rods, and subsequently zoogloea masses consisting of short rods, many containing spores, the rods being usually or frequently in pairs, with spores at their attached extremities, longer filaments composed of from four to six rods, and in some cases filaments with no obvious divisions, but containing spores at intervals.

The relation of these bacteria or bacilli to the white blood-corpuscles, which is noticed by Sanderson and Koch, is also mentioned by Birch-Hirschfeld and other observers as holding good also in the case of micrococci. They found that both in pus and blood there was a marked tendency to accumulation of these organisms in the protoplasm of leucocytes, especially around the nucleus, which in some cases were obscured by them.

It may be that the disappearance of these bodies, partial or apparently entire, from the blood in the cases observed, may be due to the tendency which leucocytes display to take up all sorts of foreign particles, a power which they share with all active protoplasm.

It is natural to inquire how it comes to pass that, in those cases in which bacteria were found in the blood during life, they were not always found in the microscopic examination of the organs after death. The discrepancy is, however, more apparent than real. For as regards bacteria of the size and appearance of bacteria termo, they were in so small a quantity in the blood that they would only be accidentally discovered in examination of sections. Moreover, in sections examined in the ordinary way, isolated bacteria would not be recognised and could not with certainty be distinguished from fibrin filaments or other minute lines. In sections examined by the special method above detailed, a few bacteria might always be seen; but so long as only isolated rods were seen, it would be impossible to decide whether they might not have been introduced from without during the examination. The presence of the characteristic movement during life is the main test of their nature; this is of course absent in sections. It is only when they occupy definite positions, are in large numbers, or are accompanied by other changes, that they can be definitely stated to exist in the sections examined.

With regard to the far smaller bacteria seen in some cases (especially that of McG—, Case 4), it appears to me very doubtful whether they are likely to be discovered after death unless by other means than those we have employed. For, owing to their extreme minuteness and tenuity, it is almost impossible to distinguish them except when in movement, and the other tissue elements, filaments of fibrin, &c., bear so close a resemblance to them that it is extremely difficult to assert their presence with assurance. It is true that Koch states that he has succeeded in identifying them in sections by means of his method and of a special illumination. We did not succeed in sufficiently destroying all the fibrin filaments in any specimen to allow of their certain recognition.

Other considerations have already been referred to, viz., the fact that these bodies only appeared in the blood from time to time, and then disappeared, indicating the probability of what Koch and others assert as a fact, that they are taken up by the leucocytes and also by other active protoplasts.

Note on the Injection of Pus.

The important series of observations made by Dr. Birch-Hirschfeld ("Arch. de Heilkunde," 1873, p. 193, *et seq.*) may here be referred to, as having an important bearing on this subject.

In the examination of healthy fresh pus from non-febrile patients he found that the pus-corpuscles were distinguished by their equal size, rounded form, sharp contour, clear protoplasm, and the fact that the nuclei were mostly invisible without the use of reagents. Together with this he found also the coarsely granular cells filled with fat granules, which are well known as occurring in old pus. In the liquor puris no bacteria were found, unless one here and there in a wound exposed to the air; and that especially where there were many suppurating wounds, as in hospitals, and where pus was retained and decomposed. In the case of a gangrenous wound in connexion with bone

disease in a phthisical patient, he also found bacteria. Frequently, however, he found micrococci in pus, especially in hospitals, and their appearance coincided with the onset of fever. The micrococci thus observed were smaller than *micrococcus ureæ*, but larger than those described by Cohn in vaccinia, and by Oertel in diphtheria. The medium length of the pairs of micrococci was from 2 to 4 μ. They had no definite movement.

The pus in which they were found was altered, the cells, instead of being of uniform size, were very various in size, their contour was not sharp, or uniform; often there appeared to be projections upon it. The protoplasm was more cloudy than natural and was filled with granules, smaller than fat-granules, and with a different refraction. The nuclei were visible without the aid of reagents. On treatment with acetic acid, instead of the nuclei becoming clear and the protoplasm swelling up and disappearing, there remained rounded granules arranged in chains, some of which soon float out. These are also insoluble in ether. Similar changes are also seen where the bacteria of ordinary putrefaction are present.

As the process advances the micrococci multiply and then colonies are formed; these being masses of micrococci, with others isolated or in pairs lying around them. Hirschfeld never saw the zoogloea masses with intermediate substance described by Cohn. As the number of these colonies increases, the pus deteriorates, and Hirschfeld believes that they then enter into the tissues.

Hirschfeld examined the *blood* also, and found that just as the local deterioration of the wound is parallel to the increase of the bacteria in the normal secretion, so do the severity and the rapid course of the infection agree with the number of the bacteria which are present in the blood.

In cases of recent amputation in which any number of micrococci were seen in the wound, similar ones were very soon seen in the blood. In no case did he find that the number in the blood bore any comparison with the number found in the wound, but towards the fatal termination he often found two or three to one red blood-corpuscle. Usually only the smallest form of double micrococci were to be found. Hirschfeld remarks that the single ones are not readily to be distinguished from isolated elements, due to breaking down of the white blood-corpuscles, but those of which he speaks resisted liquor potassæ, acetic acid, and ether. He also found changes in the white blood-corpuscles similar to those seen in pus-corpuscles, though in a less degree. He observed also a great increase in the number of the leucocytes. No change was found in the red blood-corpuscles such as that described by Coze and Feltz.

In some cases, however, he found little or none of the above-mentioned changes in the blood. Hirschfeld next made a series of experiments on animals (rabbits and dogs), with a view to ascertain the comparative effects of injection of healthy pus and pus containing micrococci and bacteria in three series of experiments.

Series I.—The injection of healthy pus produced no results beyond slight temporary fever.

Series II.—On the other hand, pus containing micrococci when injected produced fever of an irregularly intermittent type, with loss of appetite, wasting, and death in sixteen to twenty-four days. The intensity of the symptoms was proportioned to the number of micrococci, not to the condition of the patient from whom the pus was taken. On the first day no rise of temperature was observed. On the second it rose to 40·7 C., falling again to normal. On the third day, or in the night from the third to the fourth, there was a marked rise of from 42° to 43°; and from that time rapid wasting, loss of appetite, &c. Again the temperature fell, rising again to 42° or 43° before death. Some of the animals died in seven days, one lived twenty days, but death was rarely delayed beyond the fourteenth day.

Post mortem there was found suppuration around the seat of injection. When the pus was merely injected into the subcutaneous tissue, there was but little affection of the internal organs. The kidneys were usually diseased, presenting degeneration of the epithelium, sometimes colonies of micrococci in the tubes. Abscesses were found in the lower lobes of the lungs, in two cases only with colonies of micrococci in the vessels. In one case, masses of micrococci were found in the vessels in the liver with surrounding purulent

infiltration. The *spleen* was much enlarged, the cells swollen and granular, like the leucocytes in the blood.

Series III.—A third series of experiments was made with pus containing many ordinary rod-shaped bacteria of ordinary decomposition, some from a case already mentioned where they were found in the wound, others from pus left to decompose.

Some pus which contained micrococci and bacteria was taken; one half injected at once, produced results similar to those in Series II., the other half, diluted with distilled water, was placed in a glass closed with a cotton-wool plug. This latter did not develop the micrococci nor bacteria after the first day. Then he injected some of this, and in eighteen days no symptoms had appeared.

When, however, the putrid pus was injected fresh, containing bacterium termo and lineola and bacillus subtilis, the action was the same whether it was from healthy or pyæmic pus, viz., it produced the usual septicæmia.

W. S. Greenfield.

APPENDIX IV.

Notes on Microscopical Examination of Organs, together with some special Notes of Cases.

Series I.—Cases examined by Dr. Greenfield.

Case 1.* *Excision of ankle. Septic intoxication.*—E. R. A—, æt. 6. Admitted December 14th, 1877. Died February 19th, 1878. Disease of ankle; excision February 16th. Delirious at 2.30 p.m. on February 18th, vomited at 8.30 p.m. when temp. 96°; at 12 midnight temp. 98°. 19th.—At 2.30 p.m., temp. 100·4°; 3.50, temp. 104°; died.

Post-mortem examination (twenty-two hours after death). Body of very well nourished female child. Slight yellowish tint of skin, especially of face; conjunctivæ yellowish. No petechiæ on skin, no anasarca.

Recent excision wound of left ankle; vertical incisions over inner and outer malleoli. Edges of wound of deep red colour, the flaps swollen; no sign of gangrene. Lower part of shaft of tibia for about half an inch bare of periosteum, dry, very soft, and of somewhat yellowish colour. An inch above the lower end the cancellous tissue appeared quite healthy. Slight inflammation of lower end of fibula; the bone appearing quite healthy. Astragalus soft and spongy, easily cut through with a knife; cancellous tissue of intensely purple colour with small hæmorrhagic points scattered throughout, os calcis also extremely soft. Signs of intense inflammation and suppuration around the wound, but not extending more than an inch above it. Some inflammatory œdema extending half-way up the leg. Popliteal and posterior tibial arteries and veins healthy. No sign of inflammation in popliteal space. Glands enlarged, but no sign of inflammation. Other tissues of leg healthy. Knee-joint healthy. No swelling along sheath of femoral vessels; superficial femoral vein contains dark fluid blood, and appears perfectly healthy, so also common femoral. The superficial glands of the groin are distinctly swollen, with small patches of commencing suppuration.

Peritoneum healthy.

Pericardium healthy, free from ecchymosis.

Heart 3¼ oz., healthy.

Pleuræ healthy.

* Vide Appendix II., Table I., Case 3.

Lungs.—Some of the larger bronchi contain fluid resembling unaltered blood. Throughout the lower lobes of both lungs, and in upper lobe of right are numerous small points of hæmorrhagic infarction, evidently due to inhalation of blood from the bronchi.

Thymus rather large, appears healthy.

Spleen small, 1 oz. in weight, firm, healthy.

Liver weighs 15½ oz., not enlarged. Gall bladder natural, and contains dark bile. On section the liver presents a very curious appearance. The centres of the lobules are of a bright canary yellow colour; the periphery is translucent and greyish, whilst an intermediate zone is of brick-red colour. In some parts of the liver the translucent greyish zone at the periphery of the lobules is more marked. The substance of the organ is generally firm, not readily broken down. On the surface there is nothing to indicate the peculiar appearance seen on section.

Suprarenal capsules.—Medullary portion somewhat congested, otherwise natural.

Kidneys weighed 5 oz. Capsule readily separated, surface smooth, substance generally of pale colour; venæ stellatæ well-marked. Cortex, especially interpyramidal portion, swollen, of a dead white colour, free from ecchymoses.

Bladder natural, contains slightly bile-stained urine.

Stomach contains altered blood mixed with mucus: otherwise healthy.

Intestines natural throughout.

Fauces and posterior nares contain fluid blood. No ecchymosis in pharynx. Larynx natural. Some ecchymoses in submucous tissue of larger bronchi.

Brain and membranes natural.

Microscopical examination.—The following parts were examined: Granulating surface of wound, periosteum and tissues surrounding the wound, glands from popliteal space and groin, liver, kidney, spleen, lung, and thyroid. All of these organs were examined with logwood and with methyl-violet staining, and by Koch's method.

Tissues of wound, &c.—The superficial parts of wound showed but little sign of granulation; the surface was covered with an amorphous granular material, which in some parts contained small masses of micrococci. The connective tissue appeared generally swollen and infiltrated with fibrinous exudation, containing but very few leucocytes. The vessels, both in the subcutaneous tissue and beneath the periosteum, showed marked changes. In many of the capillary venules were masses of micrococci, either completely blocking up the channel, or, more commonly, adherent to the wall of the vessel and projecting into the lumen. Some of the latter seemed to be remains of a larger mass, as several separate clumps were attached near each other, as if a large plug had been washed away and had left portions sticking to the wall. (See fig. 4, Plate I.)

The small arteries in the vicinity of the wound showed very characteristically the various stages of obliterative endarteritis. The organisation appeared to be much more complete than in the similar change in the skin in Case 4, and suggested the probability that it might be a more chronic process.

The periosteum itself showed no marked change beyond considerable swelling, and in some parts distant from the wound, bodies resembling typical tubercular granulations were observed.

The *glands* from the popliteal space and the groin showed little change. No micrococci were discovered in them.

Kidney.—The following changes were observed:

1. A very peculiar condition of the epithelial lining of the convoluted tubes, analogous to that seen in the hepatic cells.

2. Multiplication of the nuclei of the endothelium of the afferent arterioles of the glomeruli; proliferation also of the muscle-cells; multiplication of the nuclei of the glomeruli; these changes precisely resembling those seen in scarlet fever.

3. Thrombi, mainly composed of leucocytes, in some of the venulæ rectæ.

4. Small tracts of interstitial exudation here and there in the cortex.

1. The condition of the epithelium, both of the convoluted tubules and the ascending tubes of Henle, closely resembled that of the liver cells. They were much swollen, so that in many parts the tubes were entirely closed. The part of the cells most affected was that nearest to the basement membrane; this

appeared to have undergone a segmentation of the protoplasm something like multiple vacuolation. The more central (*i.e.* nearest the lumen) parts of the cells, though highly granular, were not segmented, or were so to a much less degree; and from this it resulted that in the centre of the tube was a core of granular protoplasm, surrounded by a few nuclei, and between this and the wall of the tube a structure, very similar to a capillary filled with blood, the segmented protoplasm looking like polygonal cells massed together. (See fig. 2, Plate IV.)

2. The changes in the afferent arterioles and glomeruli were precisely similar to those seen in the early stages of scarlatinal nephritis. The multiplication of the muscle nuclei was especially well marked, though, of course, in a very early stage.

3. *Thrombi in venules.* Only a few of these were found, but the patches of exudation seen in other parts render it probable that similar changes existed pretty widely. The thrombi was almost entirely composed of leucocytes. No similar changes were seen in arterioles.

4. The small tracts of exudation were scattered through the cortex. They consisted merely of leucocytes accumulated in the capillaries and migrating in their vicinity. They appeared to be analogous to those seen in the liver.

5. Many of the vessels were filled with blood (normally). On examination with high powers in sections prepared by Koch's method no bacteria were found. In addition to these, amorphous fibrinous plugs were found in some of the capillaries, but no micrococcus plugs.

Liver.—In the neighbourhood of the portal spaces was found great infiltration with leucocytes, giving in some parts an appearance under a low power very similar to that seen in leucocythæmia. (See Pl. V, fig. 1.) Together with this was a very remarkable condition of the hepatic cells. These were generally swollen to such a degree as entirely to obscure the normal linear radiating arrangement of the cells, especially towards the centre of the lobules (Pl. V, fig. 3), whilst at the periphery the capillary network was more distinct, and the cells in many parts were separated from the capillaries and each other, and lay isolated in the interstices. But the most striking feature consisted in an apparent segmentation of the protoplasm, which looked as if broken up into a number of somewhat angular masses within the cell, the nucleus of which was in the majority almost entirely obscured. (Pl. V, fig. 2.) The appearance differed entirely from that seen in fatty degeneration, and no oil drops were found in any of the cells thus affected. There was also a remarkable uniformity in the distribution and extent of the affection. None of the cells showed any sign of breaking down, such as is seen in acute yellow atrophy. Many cells contained abundant granules of bile pigment.*

In some of the vessels were found amorphous plugs surrounded by leucocytes, but these did not present the usual appearance of micrococci, nor were any bacteria or micrococci distinctly observed.

Lungs.—The only change found consisted in a widely distributed bronchopneumonia, many of the air cells also contained blood.

Thyroid was perfectly healthy, no micrococci discovered.

Summary.—No micrococci or bacteria were found elsewhere than in the wound, where alone micrococci were discovered. The chief morbid change consisted in the presence of intense irritation in the liver and kidney, shown by the exudation in the portal spaces, and the peculiar change in the protoplasm of the secreting cells.

Case 2. *Pyæmia (? infective myositis); abscesses in muscles of right arm, in heart, &c.; diffuse cellulitis of left arm.*—Thos. B—, æt. 25, carman. Admitted under care of Dr. Murchison February 2nd, 1877. Died February 3rd, 8 p.m. (This case is one of such rare character that the notes are given in full.)

* The change in the cells in the central zone of the lobules of the liver has a close resemblance to an exaggeration of the normal reticulated structure of the liver-cells, which has been described by Dr. Klein in a paper published since this case was examined. ("Quart. Journ. of Micro. Science," April 1879, No. lxxiv.) From a comparison of the preparations from this case with Dr. Klein's drawings, it would seem that the morbid swelling has brought this peculiar normal structure into relief.

History.—Father dead, of dropsy, æt. 48. Mother living, æt. 49, well. Patient had an attack of "sunstroke" last August; was driving his van when he felt giddy and fell off; was sick and delirious. For three weeks was in the London Hospital, and was not well for seven weeks. A few days before the present illness commenced he had a fight, and in striking missed his aim. Present illness began on Friday, January 26th, with pains across loins, which continued till two days ago. He kept at his work, however, till January 29th, but since then has kept in bed. On January 29th and 30th he had some shivering, and has since had a good deal of perspiration. On January 31st he began to have much pain across the hypogastrium shooting up to the right hypochondrium, and, as he says, "doubling him up." On January 31st he had slight epistaxis, about a teaspoonful. Vomited last night. On night of January 29th he was very delirious. On February 1st he began to have aching pains in both arms and legs, and at the same time the arms became red and swollen.

Notes on blood by house physician.—Blood drawn in capillary tube at 12 midday, examined 1.30 p.m. White corpuscles average 1 to 30 red. Bacteria numerous, very active, 20 to 40 in each field. Numerous large inactive particles.

State on admission.—Expression anxious, countenance rather sallow, but conjunctiva white. Pulse 124, feeble, regular; resp. 50, shallow. He complains of aching pains in both arms, and also in his legs. Whole of right arm and forearm much swollen, brawny red, and tender, the inner and posterior aspects of forearm especially swollen. The left arm is also swollen similarly, but to a less degree. Both pit a little on pressure. Both calves painful, slightly swollen, and hard. Tongue slightly coated and moist, red at edges. Appetite bad; much thirst. Bowels acted yesterday. Breath a little short; no cough. Some abdominal pain, but no tenderness. Spleen not enlarged. Liver not materially enlarged. In right flank a tender point midway between ribs and crest of ilium, but no tumour or induration to be felt. Lungs and heart present nothing important.

Temperatures.—February 2nd.—2 p.m., 102·6°. 3rd.—3 a.m., 102·4°; 11 a.m., 103°; 1.15 p.m., 104·4°; 2.45 p.m., 105°; 3.45 p.m., 105°; 5.30 p.m., 105·4°; 7 p.m., 106·2°.

3rd, 10 a.m.—Restless, has not slept. Mind now clear, but wanders at times. Pulse very weak, 124, small, regular. There is still general swelling, redness, and thickening of both arms, with here and there specially elevated and thickened patches of a purple hue, changing colour like a bruise; slight herpes on lips. No enlargement of glands of neck. Lungs.—Over front of left chest and in left axilla distinct pleuritic friction; back not examined. Heart.—Action feeble, pericardial friction, especially marked near apex. Abdomen somewhat tympanitic, not tender. Spleen slightly enlarged. Urine 1032, acid, contained a trace of albumen.

No shivering, but sweating at intervals. At 5.30 p.m. vomited, and violent sickness recurred two or three times. Died at 7.30 p.m. after a severe attack of vomiting. Several small bullæ seen on thorax and back before death.

Was ordered at 1 p.m. Chloral Hyd., gr. xx; Pot. Brom., gr. xxx; Quin. Disulph. gr. v, 4tis h.

Post-mortem examination (on February 5th, forty-three hours after death. Weather cold and dry. Thermometer 30° on night of 4th and 5th. Highest temp. on 4th, 45°).—Body of strong muscular man. Rigor mortis strongly marked both in upper and lower limbs. No external marks of injury or of open wound, except a small graze on lower lip. No petechiæ on skin.

Both arms swollen; right with some superficial abrasion from desquamation of cuticle on forearm. Some rounded swellings on forearm, and one near elbow. Left arm generally swollen.

On making incisions lengthwise into right forearm some general œdematous infiltration of subcutaneous tissue and of intermuscular planes was observed. At the points of most marked swelling, circumscribed inflammation of the subjacent muscles was found, in some places extending deeply nearly to the bone, the muscle being partly injected, partly whitish, and in part broken down into a pinkish grumous pus. In other parts was a more diffuse myositis. The cellular tissue adjacent also showed signs of suppuration, but this appeared to be secondary, not running along the septa, as in diffuse cellulitis. Some

serous infiltration and general swelling extended upwards at back of arm, but little on inner side. Brachial vessels and veins apparently healthy throughout. No sign of swelling in axilla. Axillary glands slightly reddened, but free from obvious inflammation. Elbow-joint healthy. Bones of forearm and arm also apparently healthy.

Left arm: subcutaneous cellular tissue from wrist to shoulder infiltrated with serum; in some places swollen to half an inch in thickness. No sign of suppuration here. No abscesses anywhere seen in muscles.

Both knee-joints healthy. Some slight serous infiltration of cellular tissue over left calf. No sign of suppuration. Femora and left tibia and fibula healthy, so far as could be ascertained.

Fauces, mouth, pharynx, œsophagus, and larynx and trachea normal, with the exception of slight serous infiltration of muscles of pharynx and cellular tissue at posterior part.

Mediastinum normal; so also sternum, ribs, &c.

Pericardium. — Normal. Some minute subpericardial ecchymoses over anterior septal groove.

Heart.—14 oz. Right auricle much distended with imperfectly coagulated and somewhat viscid blood; so also right ventricle. Left auricle and ventricle containing very dark treacly blood. Endocardium very deeply stained everywhere. Muscular tissue of wall of left ventricle in its upper half of pale yellowish-white colour, mottled here and there with ecchymosis, the section in places granular; the condition apparently one of acute myocarditis. No actual abscess to be discovered. Lower down were one or two smaller patches of similar change. Valves normal. Entire absence of all signs of endocarditis.

Pleuræ.—Right, with general recent inflammation over lower lobe. Left, with a small patch of recent inflammation, covered by a thin layer of lymph, about one inch in diameter, over the small tongue of lung overlapping the pericardium; none elsewhere.

Lungs.—Right, 1 lb. 6 oz.; left, 1 lb. 5½ oz.; both with the general appearance of commencing decomposition, and some general congestion. One or two small recent pyæmic infarcts, only partly decolorised, in lower lobe of right. One in anterior part of left, beneath inflamed pleura above mentioned. No sign of broncho-pneumonia.

Liver.—4 lb. 1½ oz., somewhat enlarged. Blood flowing from hepatic veins, turbid, pinkish, as if mixed with milk. Opaque swollen condition, as if from commencing decomposition.

Spleen.—4 oz., small, firm.

Kidneys.—14½ oz., swollen, and opaque whitish colour of cortex. Staining from decomposition of blood.

Stomach and intestines.—Normal.

Brain and membranes.—Normal; so also sinuses and temporal bones, so far as examined. Brain weighed 2 lb. 15 oz.

Muscles in spinal groove opposite spine of scapula on left side appeared slightly injected, as if from bruising, but this somewhat doubtful. Bones of spine, membranes, and cord normal throughout.

Microscopical examination of heart.—The sections were examined by logwood staining, and also by staining with methylanilin-violet, and treatment with acetic acid. In all the sections were tracts of various size, which stained more deeply with methylanilin. In these positions were found very numerous bacteria in the intermuscular septa. In some parts they were densely packed, so as to form a distinct mass of colour visible with a low power. More commonly they were only thinly scattered, being especially abundant within and around the blood-vessels. In some of the smallest arterioles they appeared nearly to fill the lumen; in others, they lay lengthwise along the wall, within or outside it. In many parts they were found only in the arterioles or in their immediate neighbourhood, but in some they penetrated along the capillaries, and formed a network between the muscular fibrils; scarcely any penetrated the sarcolemma.

These bacteria were for the most part rod-shaped, measuring 1 μ to 3 μ in length, though many reached 4 μ, and from ·3 μ to ·5 μ in thickness. In many of the longer ones a process of fission was visible. One or two longer filaments, 12 μ in length, were seen; these contained numerous oval spores.

In some parts clumps of very minute spores were found, resembling micrococcus clumps, but these were very scanty.

Muscles of arm.—The muscular tissue in the neighbourhood of the abscesses presented extensive tracts of inflammation, exudations of leucocytes, here and there small hæmorrhages. The muscular bundles were separated in many parts by fibrinous exudation, with but few leucocytes, the fibres having in many places lost their striation and assumed a peculiar glassy appearance.

On examination with higher powers, only one or two bacteria were found on very prolonged investigation of numerous sections. But both in the blood in the vessels, in thrombi, and in the exudation, were seen, either isolated or in clusters, micrococci, measuring from 2 to 4 μ in diameter, mostly rounded, a few ovoid. In some of the sections, in which the colour had been very completely removed from the other tissues, these came out with great distinctness. Larger masses of micrococci were also discovered in some parts, resembling those seen in the other cases (see fig. 1, Pl. II.).

Skin over inflamed muscles presented only scattered patches of inflammation, in which were no special characters. No micrococci, or bacteria discovered.

Kidneys.—Nothing abnormal observed.

Lungs.—Slight catarrhal exudation in many alveoli; no other specific change.

In connexion with this remarkable case, some similar cases may with advantage be mentioned, which are recorded by Dr. Nicaise, under the title of *infective myositis* or *malignant myositis*, in the "Revue Mensuelle de Médecine et de Chirurgie," 1877.

He states that the disease consists of acute, diffuse, suppurative myosotis, accompanied by grave general symptoms and rapid death.

Case a.—M. X—, æt. 40. Epileptic. July 5th.—Had a fit. Worked the next day.

6th.—Pain in the inner and upper part of the thigh. Could not work.

8th.—Saw a doctor. Deep-seated inflammation of thigh.

10th.—Fever. Inflammation of muscles of thigh, supposed to be due to rupture during the fit. Same evening had pain in right shoulder and side.

11th.—Side of chest swollen, œdematous, skin thickened and hard; yellowish tint, some redness; redness of thigh; high fever, sweating.

12th.—Œdema and redness extended; delirium. Temp. 39·4° (103° F.).

13th.—More swelling; no fluctuation. Some petechiæ on back. Temp. morning, 38° (99·5° F.); evening, 104°.

14th.—No fluctuation, not much change; sweating; no albumen in urine.

15th.—Worse. Temp. 104°. Died July 15th, night.

Post-mortem, July 17th.—Decomposition far advanced. *Thigh.*—Subcutaneous cellular tissue contained no pus, adductor muscles infiltrated, no collection of pus; muscular substance greyish, infiltrated with pus, spongy; some infiltration in pectoralis major and serratus magnus; congestion of lungs; spleen black and pulpy; liver fatty; kidneys healthy; no superficial wound could be found anywhere.

Case b. *Suppurative myositis of triceps femoris.*—Man, æt. 45, butcher, strong; following some hard work, he felt pains in the thigh, and was obliged to keep his bed. On admission a few days after there was a large yellowish-red patch on the thigh, indurated and moderately sharply defined. No subcutaneous collection of pus; doubtful deep fluctuation; general state bad. The following day there was evident fluctuation, an incision made. The triceps was found to be infiltrated with pus; a purulent layer superficial to it. Abundant serous infiltration. The man died four days after. Post-mortem not recorded.

Case c. *Varicose ulcer; lymphangitis; purulent arthritis of knee; diffuse myositis of triceps femoralis and brachialis.*—M. X—, æt. 62. Good previous history. Simple fracture nine years before. Two years afterwards a varicose ulcer formed. On admission there was a large foul ulcer on the *left* leg. Viscera healthy. Washed with carbolic acid, 1 to 50.

A few days (October 3rd) after admission a severe rigor and lymphangitis, with swelling of the inguinal gland, appeared.

4th.—Worse.

6th.—Pain in *right* knee.

7th.—Worse.
8th.—Worse.
9th.—Incisions into knee.
10th.—He died.

Post mortem.—Serous infiltration of thigh. A few abscesses size of peas in course of the lymphatics. Vastus internus infiltrated with pus. Adductors pale and discolored. Purulent infiltration of the triceps of the arm. Elbow healthy. Hypostatic congestion of lungs. Nothing in heart or brain. Liver yellow and a little soft. Spleen blackish and diffluent. Kidneys congested.

Nicaiase wisely offers no explanation of these cases.

Case 3.—*Chronic pyæmia with multiple suppuration.* (Case in Appendix II.; Case 11 in Appendix III.) — E—. Died July 14th, 10 a.m. Post mortem 2.30 p.m.

Post mortem (four hours and a half after death) by Dr. Greenfield. Weather warm, very wet. Wind south-west.

Rigor mortis scarcely commenced. Body generally emaciated. Some anasarca of both feet and ankles. No petechiæ or ecchymoses. Some swelling of tissues below right clavicle and in sub-pectoral region. Blood in veins thin and watery.

Forehead, eyelids, especially left, upper part of nose, and front of scalp of dusky red colour, swollen, with numerous groups of small pustules scattered over it, some broken, covered with thin watery secretion or scab, but many, size of small shot, embedded in substance of swollen and inflamed tissue. On cutting into this the whole swelling found due to similar multiple points of suppuration about the size of mustard seed or rather larger. Small, dusky-red patches in skin over left tibia; on section these have exactly the same appearance as those on forehead. Left little finger, also, especially along middle phalanx, presents an exactly similar condition going down to bone. Other patches not examined. Both ankles, both knees, and left elbow, with shoulder-joints, examined. No pus in any of them, only some excess of synovia, especially in ankle and right knee. Caries of third metatarsal bone of right foot. The whole upper lip completely destroyed—eaten away—up to root of nose. The alveolar border of jaw exposed and slightly invaded. Edges of ulceration rather smoothly cut, covered with thin secretion. Glands on left side of neck in upper part, at one point adherent to the skin, and converted into thick pus. Some ulceration of skin not unlike strumous ulceration. No obvious recent enlargement of glands in upper part of neck. Swelling in lower part of neck on right side, just above clavicle and shoulder; and much more marked just below clavicle. Considerable œdema of subcutaneous cellular tissue. In deltoid and pectoralis, close to clavicle, and for about two inches below, signs of intense inflammation, and very numerous scattered points of suppuration, the pus being very thick and almost semicaseous—the condition exactly like that in skin, &c. A vein of some size here found filled with soft pinkish-white clot. Axillary vein not affected. No obvious swelling of glands. No suppuration.

Brain.—Membranes, &c. of brain quite normal. Brain remarkably healthy looking.

Pericardium contained about two ounces clear serum.

Heart large, healthy looking. Valves, &c. normal.

Pleura each contain nearly a pint of clear serum, a coagulum of lymph in left.

Both *lungs* spotted with numerous ecchymoses. On surface very abundant, scattered, pale nodules, varying in size from a small pea to half an inch in diameter, somewhat prominent, the larger ones flattened on surface, irregularly rounded; all surrounded by a more or less marked zone of congestion and minute ecchymoses, but no exudation. Patches of collapse scattered over posterior surface of both. On section of lungs patches similar to those seen on surface were found also to be scattered irregularly through both lungs, most marked towards surface. They were of yellowish or pinkish-white colour, rather sharply outlined, surrounded by a zone of congestion, which in some was very intense; they had an almost caseous appearance, and seemed to be made up of multiple points of suppuration. A few were softened and tending to excavate. Towards the root of the lung, and extending from it in arbo-

rescent fashion, very beautifully defined patches of broncho-pneumonically arranged fibroid tubercle, in one or two places beginning to cascate, but these quite exceptional.

Bronchial glands not obviously affected.

Peritoneum normal.

Liver rather large and pale, otherwise normal.

Spleen enlarged and deeply congested, but slightly mottled, rather firm and dry. No infarcts.

Stomach normal.

Intestines not fully examined. Mesenteric glands apparently normal.

Kidneys rather large and pale. Some general swelling. Nothing else.

Bladder not examined.

In left groin a puckered smooth scar as from old abscess. Around this a dusky purple blush and slight central sloughing. On section the superficial layers of skin separated by pus from deeper, and in tissues around are multiple points of suppuration like those elsewhere. One small pustule on glans penis.

Larynx.—Slight thickening about ary-epiglottic fold; otherwise normal.

Trachea normal. Perforating ulcer of soft palate with thickened edges, quiescent, chronic.

Tongue coated and thick.

Microscopical examination. Finger.—In the part of the finger examined were numerous minute abscesses, measuring from 1 millimètre to much smaller; these were mainly situated in the skin and subcutaneous tissue. For the most part they appeared to have been formed around veins, for some of the smaller abscesses occupied the situation of a vein, part of the coats of which were still visible, the abscess apparently consisting merely of a vein which had contained a puriform clot, its walls infiltrated with leucocytes, and more or less broken down. These abscesses contained broken-down pus, together with a large number of ovoid or rounded bodies, which stained very deeply with logwood or methylanilin violet (Pl. IV. fig. 1).

The smaller arteries in the neighbourhood of these minutes abscesses had undergone a change analogous to that which has been described as "obliterative endartiritis." The outer coat, where actual suppuration had occurred in its vicinity, was usually much thickened and infiltrated with leucocytes. The middle coat showed little or no alteration. The inner coat was swollen somewhat irregularly, in some part to such a degree as nearly to exclude the vessel, in other parts the lumen was occupied by a thrombus. The swelling, though mainly hyaline, was here and there infiltrated with leucocytes. (Fig. 3, Pl. III.)

The endothelium was markedly proliferated in some of the sections, appearing mainly to compose the plug in the interior of the vessel, and in all the sections it had become desquamated.

One or two bacteria and micrococci were found in the blood, in vessels and elsewhere, but not in sufficient numbers or with sufficient constancy to appear to be of any importance.

Muscles.—The muscles in various parts contained also numerous minute abscesses, scarcely larger than those in subcutaneous tissue. Their contents resembled those in the skin; surrounding them were somewhat extensive tracts of fibrinous exudation, of blood, and of leucocytes. The muscular fibres presented various stages of degeneration. No bacteria or micrococci anywhere seen.

Lungs.—The portions examined showed extensive pneumonic changes and tuberculosis, but nowere any sign of bacteria or micrococci.

Kidneys.—Nothing abnormal.

Case 4. (Case 4 in Appendix III.)—McG—. Clot *in situ* in femoral vein, lung liver, and kidney examined; sections stained with logwood, methylanilin, violet, and the modified method of Koch.

Thrombus in situ.—Nothing special was observed either in the clot itself or in the walls of the vein. Only a small portion of the clot remained adherent

to the wall; this showed no sign of organisation. No bacteria or micrococci, nor anything beyond what is usually seen.

Lungs. — Scattered patches of pneumonia; some of alveoli filled with catarrhal, others with fibrinous exudation, but with no special features. Many vessels contain recent thrombi; in some of these bodies resembling micrococci seen, but no bacteria, and no clumps of micrococci.

Liver for the most part natural, but some of nuclei of hepatic cells stain very deeply with logwood or methylanilin, and with a high power ($\frac{1}{16}$th) this appears to be due to the accumulation of a large number of very deeply stained bodies resembling micrococci completely obscuring the nucleus; in some as many as twenty or more counted; in others only two or three seen; others quite natural. One or two small vessels thrombosed; thrombi contain much granular matter, scarcely stained.

Kidney with a low power, no very marked change beyond very early stage of chronic interstitial nephritis in some of superficial parts of cortex. Marked cloudy swelling of epithelium of convoluted tubes. No micrococci or bacteria.

Case 5. *Acute necrosis.* (Case 17 in Appendix III.)—M. A. A—.

Skin.—The skin of the finger contains here and there minute abscesses, chiefly in subcutaneous tissue, not more than half millimètre in width. They are sharply defined, contain only leucocytes, broken down in centre. In the neighbourhood of these abscesses micrococci are found scattered through the tissue, some isolated, many others double, a few in lines or clusters. No clumps or zoologœa masses in vessels. Some of the arterioles are filled with thrombi, consisting almost entirely of fibrin and leucocytes; and some misrococci are observed in them, scattered here and there.

Brain.—Scattered through the more superficial parts of the brain are found here and there minute abscesses (Pl. V., fig. 5) and points of infiltration with leucocytes which have not yet broken down. Numerous minute arterioles plugged with leucocytes, some of these leading to the parts infiltrated with leucocytes. Micrococci were found in these infiltrated portions.

Heart.—Pericardium here and there swollen and infiltrated with leucocytes, especially around vessels; many small vessels filled with plugs, leucocytes, and fibrin. Near the surface numerous arterioles filled with clumps of micrococci, measuring from 6 to 15 μ in diameter; others contain only smaller masses not completely filling the vessels; some smaller clumps also seen in the tissues, not obviously in vessels. The individual micrococci measure about ·15 to ·3 μ in diameter. (Pl. II., fig. 2, and Pl. III., fig. 1.)

Many small vessels in superficial parts of heart are filled with leucocytes and fibrin, and some micrococci are found here and there in the vessels.

Lungs.— Nothing of any importance was found in the lungs in parts examined.

Thyroid body.—Scattered through the thyroid were numerous points of suppuration, varying in size from the minutest speck to the size of a mustard seed. On microscopic examination many other foci of commencing inflammation are found. In the larger abscesses, and those completely broken down, nothing special is observed, but in several of those not yet broken down there are seen clumps of micrococci near the borders of the inflamed tract. None were found in the vessels elsewhere in any of the sections examined.

Supra-renal body.—In one of the sections an arteriole plugged with micrococci was found, but none in any other section; and no other changes.

Case 6. (Case 5 in Appendix III.)—H—. Liver and kidney alone examined by logwood and methylanilin methods.

Liver.— Hepatic cells not obviously affected. Slight interstitial fibrosis at periphery of lobules; this very probably old. In one of the small branches of hepatic vein a thrombus was found composed almost entirely of leucocytes. No bacteria or micrococci.

Kidney.—Some general chronic interstitial change, rendering difficult any recognition of acute interstitial changes. Epithelium of convoluted tubules in many parts swollen, very granular, and breaking down. Hyaline, colloid, and epithelial casts in small number in straight tubes. No bacteria or micrococci discovered.

Case 7. *Amputation of left thigh for disease of knee-joint; suppuration in shoulder-joint; pyæmia.*—Jane H—, æt. 36.

Post-mortem examination.—Flap healthy looking, but rather deeply undermined. Lower half inch of shaft of bone bare, but not necrosed. No osteomyelitis. No general cellulitis. Sheath of femoral vessels through its entire length thickened, infiltrated, but free from pus. Superficial femoral through its whole length filled with creamy fluid, and small shreds of soft, creamy, pinkish clot, its walls much thickened, especially inner coat, which is of greenish-yellow colour, and rough on its interior surface, injected here and there. At the lower end, vessels filled by firmish adherent clot, and completely closed. The softened condition of the clot extended as far as the first inch of the external iliac vein, which beyond this was completely closed by a rather firm dark red clot adherent to its walls; beyond this the vessel channel again became free, and its walls were quite healthy. Suppuration in right shoulder-joint only; all the other joints of limbs healthy. Hypostatic congestion of lungs, with a few small patches of bronco-pneumonia in lower lobes. Spleen 8 oz., somewhat enlarged, firm. Nothing special in other organs.

Microscopic examination of liver and kidney.—*Liver* fatty, with some interlobular fibrosis resembling that in Case 1.

Kidney, no special change. No micrococci or bacteria were discovered in either.

Case 8.* *Morbus coxæ; lardaceous degeneration; pyæmic abscess in liver. Liver.*—Presented very advanced lardaceous degeneration, together with some chronic interstitial changes. Minute abscesses were scattered through its substance. No special changes, however, were discovered.

Case 9. *Amputation of thigh.*—*Liver* fatty. Some of the liver-cells present a very peculiar deep staining with hæmatoxylin. In the capillaries adjoining are seen what appear to be double micrococci in considerable number, but not forming anywhere zoogloea masses. No other changes of importance.

Case 10.† *Phlebitis of portal vein; abscesses in liver; pyæmia.*

Portal vein and clot.—Nothing special was found either in the portal vein and the tissues surrounding it, or in the clot *in sitû*. Both presented the usual appearances. No bacteria or micrococci were found in either.

Kidney presented nothing special beyond some swelling of epithelium of convoluted tubules.

Case 11. *Stump from case of pyæmia following amputation of thigh for compound fracture.*—A section about ¼ inch in depth to include the granulating surface of the wound and the subjacent tissues was examined by logwood staining and Koch's method.

The granulating surface appeared healthy; the granulation cells for the most part of normal size and appearance. Towards the surface of the wound micrococci separate or in dumb-bells were seen in some abundance, but not extending more than a very short distance into the granulations, except here and there where they penetrated much more deeply.

The fatty and connective-tissue planes were for the most part natural, and contained no fibrinous exudation. But here and there the connective-tissue corpuscles were found to contain large numbers of micrococci in their protoplasm, and micrococci also lay scattered around those cells where they infiltrated them in considerable number. The contrast between these cells and healthy ones was very striking, especially in sections stained with methylaniline, and only partially cleared with acetic acid.

Blood contained in some of the vessels contained numerous micrococci, which appeared also to infiltrate the endothelial cells. Some of the arterioles showed a thickening of their intima, but they were for the most part natural. Some veins showed infiltration of their walls with leucocytes, which also filled the lumen, but beyond this nothing abnormal.

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* Appendix II., Table III., Group I. (B) Case 6.
† Appendix II., Table VI., Group IV., Case 6.

Case 12.* *Ulcerative endocarditis.* (No. 18 in Appendix III.)—Only the spleen and kidney were examined.

Spleen.—Tissue for the most part natural, but here and there were masses of micrococci. They could not always be seen to be surrounded by the wall of a vessel, but in other respects were similar to those seen in other cases. (Pl. I., fig. 5.)

Kidney.—No micrococci discovered. Here and there were small atrophied tracts, apparently due to old infarction, but no recent change was discovered.

Series II.—Cases examined by Mr. McCarthy.

Case 13. *Pyæmia.* Appendix II., Table II., Group I. (*a*), Case 13.

Wound.—Surface covered with a thick granular layer which stained with logwood. It was probably composed of sloughy tissue containing numerous micrococci. No micrococci in the vessels or lymph spaces beneath.

Secondary subcutaneous inflammation.—Presented nothing characteristic. No organisms to be detected in a section stained in the ordinary way with logwood.

Lungs, liver, spleen, kidneys, and brain also examined. Nothing characteristic to be observed. In one of the lumbar lymphatic glands a single colony of micrococci was observed in a vessel. With this exception the results of the examination of about fifty different slides were purely negative.

Case 14. *Pyæmia.* Appendix II., Table II., Group I. (*a*), Case 5.—An examination of about fifty slides from the heart, liver, lungs, spleen, kidneys, brain, and thyroid body showed nothing in the way of colonies of micrococci, or of bacteria, or microscopic points of inflammation or suppuration.

Case 15.—Victor G—, æt. 2, died October 20th, 1877. Appendix II., Table IV., Case 3.

Lungs.—Some of the smaller blood-vessels plugged with infarcts. Extravasation of blood into air vesicles in neighbourhood of such vessels. In other parts catarrhal pneumonia, and among the cells filling the air vesicles of such parts some multinucleated cells and groups of micrococci. Small lymph follicles in walls of bronchi.

Thyroid gland.—Some of the larger veins plugged with coagula, in which are groups of micrococci. Other vessels filled with débris of disintegrating blood-corpuscles.

Heart.—Subpericardial vessels filled with blood. Thickening of pericardium in parts, due to swelling of bundles of fibrous tissue. A large mass of micrococci in one specimen, with commencing granular disintegration of surrounding muscle fibres. The mass of micrococci had in part escaped from containing vessel through a rupture of the walls, and was surrounded by some few pus-corpuscles.

Spleen.—In perfect preservation, and numerous sections showed nothing abnormal.

Liver.—Many capillaries plugged with micrococci, and in some parts, and these not always near the plugged capillaries, liver-cells were infiltrated with fat, and in some cases to such an extent that the cells after treatment with alcohol and oil of cloves resembled ordinary fat-cells after like treatment.

Kidney.—Glomeruli of Malpighian bodies obscured by increased number of nuclei. Thickening of coats of afferent vessels. Epithelium of convoluted tubes swollen so as in many parts to completely obliterate lumen of tubules. Many vessels between collecting tubes plugged with micrococci, and in some parts such groups of micrococci formed centres of miliary abscesses. In some vessels fibrinous infarcts and extravasated blood in neighbourhood. In many collecting tubes small mulberry masses, composed of aggregations of small bodies, usually round, but sometimes irregular in shape from mutual pressure. These small bodies, usually about one third the size of a coloured blood-corpuscle, stained deeply with hæmatoxylin at periphery, and remained colourless at centre, and dissolved without effervescence in dilute hydrochloric acid.

* Appendix II. Table X., Group VIII., Case 1.

Supra-renal capsules.—Blood-vessels in medullary part plugged with coagula containing groups of micrococci.

Brain.—Except an unusual number of so-called corpora amylacea, nothing noticeable.

CASE 16.* September 18th, 1878.

Lungs.—Enormous quantities of micrococci, sometimes with a few intermixed blood-corpuscles, filling large subpleural vessels, sometimes in perivascular sheaths of large blood-vessels. In one specimen a small branch given off from a large vein had burst, and through the rupture blood had become extravasated into surrounding structures, and in this were large groups of micrococci (Plate XI., Fig. 1). Throughout substance of lung many capillaries on air-vesicles were plugged with these organisms, which were also to be found mingled with the disintegrating cells in patches of catarrhal pneumonia. The surface of pleura was covered with fibrinous vegetations, and most especially where subjacent vessels were filled with micrococci. In many parts also much thickened by swelling of bundles of fibrous tissue.

Liver.—Connective tissue in portal canals much swollen, and in many parts crowded with colourless blood-corpuscles. Epithelium of hepatic ducts swollen so as often to almost obliterate lumen of duct. Some of the portal veins plugged with fibrin. Liver acini much changed. In some, hepatic cells swollen and cloudy; in others, the cells shrivelled, with nuclei much diminished in size or absent, and containing a great abundance of bright yellow pigment granules. In the latter kind the sustentacular structure of the acinus was much thickened, which was especially noticeable where the loosened hepatic cells had fallen out, leaving exposed to view the containing network. In this sustentacular structure were many connective-tissue corpuscles, some multinucleate. In a section a single acinus might be found with greatly shrivelled cells completely surrounded by acini, which differed from the normal only by cloudiness of the hepatic cells. In other parts of the same section the shrivelling process had extended through contiguous acini as to leave a comparatively normal acinus insulated. In every section capillaries could be found plugged with micrococci, often, from unequal distension, presenting a varicose appearance. Such capillaries were to be found in slightly changed acini, as well as those which were most altered from the normal.

Kidney.—Sections from this organ presented very different appearances at different parts. In some places the epithelium was normal and well preserved; in other parts of the same section it was swollen, granular, and extensively detached from the basement membrane, which in parts where the loosened epithelium had fallen out appeared thicker than usual. Many of the collecting tubules contained mulberry masses, such as were described in Case 15. In other tubules the epithelium was much pigmented. The vessels varied in appearance like the tubules. Some were normal and well preserved; in others the endothelium was much detached, and either lay as a collapsed tube in the lumen of the vessel, or had its component cells separated from one another and intermixed with blood-corpuscles. In some of the glomeruli there was an increased number of nuclei; in others the walls of the component vessels were swollen and stained deeply with hæmatoxylin. Of this latter change many different stages could be observed in the same specimen, from slight swelling of some of the component vessels to complete obliteration of the lumen of all, and the transformation of the glomerulus into a homogenous deeply-stained mass. The afferent vessels were similarly affected, and all the structures in their walls seemed to participate in this change, but the adventitia most. Some of the small arteries contained apparently fibrinous embolisms. Plate VII., Fig. 3, shows such an embolus, lodged at the point where the vessel bifurcates. There was little change in immediate neighbourhood, and it had probably become lodged there just before death. Some few vessels contained groups of micrococci, but in this respect the sections of kidney presented a marked contrast to those of the liver and lung from the same subject. Sections of brain, testicle, and of a large vein filled with a thrombus were examined, but nothing noteworthy was observed. (No other viscera sent for examination.)

* Appendix II., Table VII., Group V., Case 2.

Case 17. *Lithotomy.* Group I. (*a*). Case 14.—*Viscera examined*—Heart, lung, lymphatic glands, liver, kidney, prostate gland. The only noteworthy change observed in this case was greatly increased cell growth in the connective tissue of the various organs except the heart. It was specially noticeable in the prostate and in the kidney. In the latter the glomeruli were sometimes completely masked by nuclei, and in some parts the intertubular structures crowded with small round cells to such a degree as to completely conceal the tubules. In many parts were also extravasations of blood.

In the *lungs* catarrhal pneumonia, hæmorrhage into pulmonary vesicles, and greatly increased lymphoid growths along bronchial tubes, were observed.

The *heart* appeared normal.

The *liver* was not well preserved. No micrococci in any organ.

Case 18.* *Infective endocarditis.* Middlesex, September 1878.—Viscera examined—Heart, kidney, liver, prostate gland.

Heart.—The subendocardial vessels of the mitral valve contained masses of micrococci. The blood-vessels showed considerable hypertrophy of the intima. This could be seen in all stages. In some vessels the intima was irregularly hypertrophied, so as to project at certain points into the lumen of the vessel. In others the change had occurred uniformly all over the intima, and this in various degrees even to complete obliteration of the lumen. The pericardial surface of the heart was in some places coated with an uniform layer of micrococci imbedded in a homogeneous substance, and in parts, where the pericardium appeared as if eroded, masses of micrococci had penetrated among the deeper structures. In some of the larger vessels in the substance of the heart were numbers of colourless corpuscles imbedded in fibrin and adherent to the wall of the vessel.

Liver.—Some of the larger vessels contained coagulated blood, with masses of micrococci imbedded in it.

There were some inflammatory changes in the connective tissue of the portal canals, but the liver-cells did not present any abnormality.

Kidney.—Epithelium extensively detached, basement membrane thickened, glomeruli with great increase of nuclei, and in some parts intertubular structures filled with round cells. No micrococci were found. Prostate normal.

Case 19. *Acute necrosis of left tibia* (Appendix II., Table IX., Group VIII., Case 4).—January 18th, 1878. Organs examined—Lungs, heart, thyroid gland, liver, kidney, testicle, and thrombosed femoral vein.

Lungs.—Catarrhal pneumonia. Infarcts with extravasation of blood. Some large vessels with fibrinous masses containing enormous numbers of colourless corpuscles adherent to a part of the circumference. Many of the pulmonary vesicles filled with degenerating epithelium, containing groups of micrococci.

Heart apparently normal.

Thyroid gland,—Micrococci in some capillaries.

Liver.—Some few groups of micrococci in large vessels. Changes in connective tissue and liver-cells, similar to those in Case 16.

Kidney.—Epithelium swollen and granular. Some glomeruli shrivelled, with thickening of coats of afferent vessels.

Testicle normal.

Thrombosed vein.—Wall of vein much thickened by swelling of fibrous tissue; vasa vasorum distended with blood. The clot which had filled the vessel had fallen out of the sections, leaving some adherent to the inner surface. This consisted of disintegrating corpuscles, fibrin, and groups of micrococci.

Lymphatic glands.—Considerable thickening of wall of vessels.

Case 20. (Appendix II., Table III., Case 16.)—Organs examined—Lungs, thyroid gland, supra-renal capsule, kidney, liver, spermatic cord.

Lungs.—Lobular pneumonia. In some of the less altered parts capillary vessels plugged with micrococci.

Thyroid gland.—Blood-vessels generally much changed. In some the intima was hypertrophied, in others the middle coat presented a homogeneous appearance stained pale violet with hæmatoxylin; in others the adventitia was much

* Appendix II., Table X., Group VIII., Case 2.

Description of Plate VI.

For the drawings in Plates VI--XI, I am indebted to the kindness of my colleague, Mr. F. Treves (J. McCarthy).

Fig. 1. From case 22, App. IV. Hartnack, ob. 7, oc. 3. Part of section of a lymphatic gland. The large vessel to the left contains a large mass of micrococci. The nuclei of the endothelial cells are swollen and granular; the lymph corpuscles round the vessel are cloudy and indistinct, from swelling of sustentacular tissue. The vessel to the right and lower part of field shows considerable hyaline changes in its walls.

Fig. 2. From same case. Hartnack. ob. 7, oc. 3. Section of a lymphatic gland, showing two groups of micrococci. That to the left completely fills a vessel and its branches; that to the right has a limiting wall only at the lower part, having elsewhere spread beyond the vessel into surrounding tissues. At the upper part the gland-substance has been replaced by a mass of fibrous tissue, with numerous clefts lined by endothelial cells. Many of the blood-vessels show great thickening of their walls, with hyaline and fibrous changes.

Description of Plate VII.

Fig. 1. (From Case 22, Appendix IV.) Hartnack, ob. 7, oc. 3. Section of lymphatic gland, showing giant-cells, swelling of sustentacular tissue, thickening of walls of vessels, and at the lower part a blood-vessel filled with micrococci.

Fig. 2. From same case. Hartnack, ob. 7, oc. 3. Section of lymphatic gland, showing commencing changes in sustentacular tissue. Swelling, with hyaline change in walls of vessels, and two groups of micrococci at upper part of field.

Fig. 3. From Case 16, App. IV. Hartnack, ob. 7, oc. 3. A small renal artery plugged at point of bifurcation by an embolus, chiefly composed of fibrin.

Fig. 1.

Fig. 2.

Fig. 3.

Dangerfield. Lith. 22 Bedford St. Covent Garden.

Description of Plate VIII.

Figs. 1, 2. From Case 21, App. IV. Hartnack, ob. 7, oc. 3. Sections of heart showing superficial rupture of muscle fibres. Fig. 1 represents a more advanced stage, and at the lower part the fibres are much disintegrated.

Fig. 3. From same case. Hartnack, ob. 7, oc. 3. Section of heart showing a vessel plugged with micrococci, which above have spread beyond the vessel over adjacent parts. The muscle fibres are quite normal.

Fig. 4. From Case 18, App. IV. Hartnack, ob. 9, oc. 3. A transverse section of a blood-vessel in the mitral valve, in which the lumen is considerably encroached upon by what is probably an irregular hypertrophy of, and change in, the *tunica intima*.

Description of Plate IX.

Fig. 1. From Case 15, App. IV. Hartnack, ob. 4, oc. 3. Section of kidney, showing a vessel and its branches plugged with micrococci and the surrounding structures infiltrated with pus corpuscles.

Fig. 2. From Case 20, App. IV. Hartnack, ob. 9, oc. 3. Section of kidney, showing relation of basement membrane of collecting renal tubules to epithelium. At the upper part is seen in profile the wall of a tubule from which the epithelium has completely disappeared, leaving the processes of the membrane which project between them distinctly visible. Below is a surface view of a similar tubule in which these processes pass completely across tubule. To the left are many stellate cells in the wall of the tubule.

Fig. 3. From Case 24, App. IV. Hartnack, ob. 8, oc. 3. Section of kidney, showing similar condition. The processes, however, are much swollen, fibrous, and stain deeply with hæmatoxylin.

Fig. 4. From Case 28, App. IV. Hartnack, ob. 8, oc. 3. Section of kidney, showing similar condition of the basement membrane in the cortical part. From two convoluted tubules the epithelium has disappeared, leaving visible these processes of basement membrane which are greatly swollen.

Fig. 1.

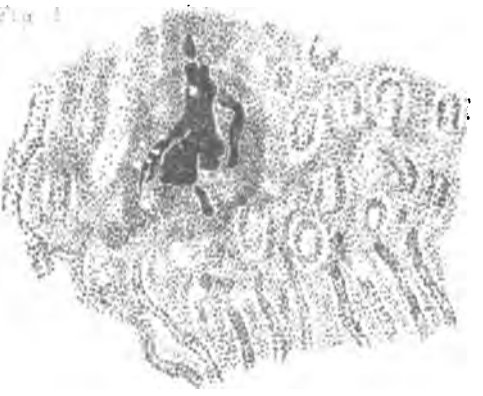


Fig. 2.

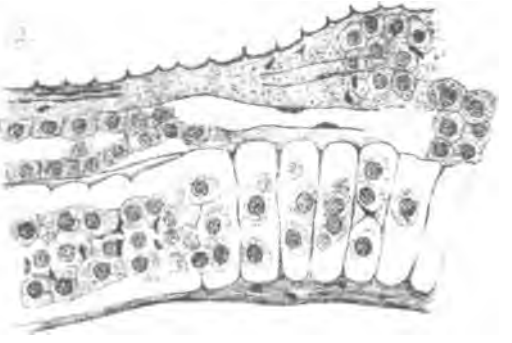


Fig. 3.

Fig. 4.

Fig. 1

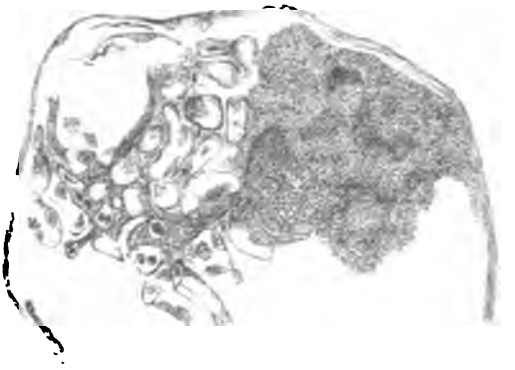


Fig. 2

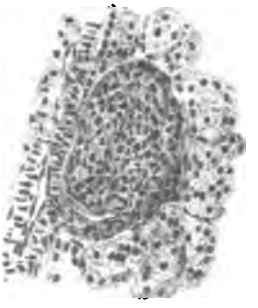
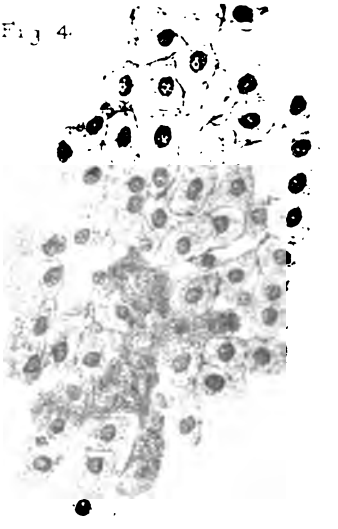


Fig. 3

Fig. 4.



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